
MEDICO-CHIRURGICAL SOCIETY
EDINBURGH

THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY

OF EDINBURGH

VOL. XVII.—NEW SERIES

SESSION 1897-98

EDINBURGH: WILLIAM F. CLAY
PUBLISHER TO THE SOCIETY
1898

PREFACE

THE present Volume is the *Seventeenth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

WILLIAM CRAIG,
Editor.

October 1898.

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	Dr R. J. Johnston, 1 Buccleuch Place,	1897
105	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens, *Treasurer*,	1883
	Dr C. Kennedy, 5 Salisbury Road,	1886
	Dr C. B. Ker, City Fever Hospital,	1894
	Dr George Kerr, 6 St Colme Street,	1885
110	Dr J. H. A. Laing, 11 Melville Street,	1889
	Dr R. F. C. Leith, 20 Merchiston Terrace,	1886
	Dr Linton, 60 George Square,	1863
	Dr Harvey Littlejohn, 11 Stafford Street,	1889
	Professor Sir Henry D. Littlejohn, 24 Royal Circus,	1853
115	Dr R. A. Lundie, 55A Grange Road,	1883
	Dr W. Tasker Lundie, 3 Glengyle Terrace,	1896
	Dr Donald Macaulay, Jumpers' Gold Mining Co., P.O. Box 1043, Johannesburg,	1895
	Dr P. M'Bride, 16 Chester Street,	1879
	Dr W. A. M'Cutchan, County and City Asylum, Burghill, Liverpool,	1897
120	Dr William C. M'Ewan, Prestonpans,	1892
	John M'Gibbon, Esq., 55 Queen Street,	1868
	Dr MacGillivray, 15 Charlotte Square,	1877
	Dr G. Mackay, 74 Bruntsfield Place,	1878
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
125	Dr Robert Mackenzie, Napier Villa, Merchiston,	1893
	Dr W. Leslie Mackenzie, 4 Summerside Place, Leith,	1895
	Dr William A. Mackintosh, 13 Abercromby Place, Stirling,	1896
	Professor Sir Douglas Maclagan, 28 Heriot Row,	1834
	Dr J. Shaw M'Laren, 14 Walker Street,	1887
130	Dr P. Hume Maclaren, 1 Drumsheugh Gardens,	1868
	Dr John Macmillan, 27 Warrender Park Road,	1897
	Dr John MacRae, Lynwood, Murrayfield,	1893
	Dr D. Menzies, 20 Rutland Square,	1878
	Dr James Middlemass, Royal Asylum, Morningside,	1895
135	Dr A. Miles, 1 George Square,	1892
	Dr J. Mill, 26 N. Fort Street, Leith,	1886
	A. G. Miller, Esq., 7 Coates Crescent,	1867
	Dr W. H. Miller, 51 Northumberland Street,	1895
	Dr D. Milligan, 11 Palmerston Place,	1885
140	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr Moir, 52 Castle Street,	1836
	Dr James Mowat, 1 Priestfield Road,	1893
	Dr John Mowat, 6 Buccleuch Place,	1885
	Professor Robert Muir, 20 Hartington Place,	1894
145	Dr Claud Muirhead, 30 Charlotte Square,	1866
	Dr R. Milne Murray, 11 Chester Street,	1884
	Dr P. Orphoot, 113 George Street,	1865
	Dr John Orr, 1 Rillbank Crescent,	1895
	Dr W. Basil Orr, 13 Braid Road,	1893
150	Dr G. Keppie Paterson, 19 Albany Street,	1889
	Dr D. Noel Paton, 22 Lynedoch Place,	1885
	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Dr R. W. Philip, 45 Charlotte Square,	1883
	Dr Playfair, 5 Melville Crescent,	1874
155	Dr T. Proudfoot, 30 Lauriston Place,	1889
	Dr Harry Rainy, 25 George Square,	1896
	Dr Rattray, Portobello,	1874
	Dr James Ritchie, 22 Charlotte Square,	1873
	Dr R. Peel Ritchie, 1 Melville Crescent,	1862
160	Dr Argyll Robertson, 18 Charlotte Square,	1861

		Date of Admission.
	Dr William F. Robertson, Charterhall Road,	1893
	Dr W. G. Aitchison Robertson, 26 Minto Street,	1892
	Dr T. R. Ronaldson, 8 Charlotte Square,	1877
	Dr R. M. Ronaldson, 17 Morningside Road,	1895
165	Dr William Russell, 3 Walker Street,	1884
	Professor Rutherford, 14 Douglas Crescent,	1866
	Dr E. G. Salt, 50 George Square,	1895
	Dr James Scott, 43 Minto Street,	1895
	Dr Andrew Semple, 10 Forres Street,	1883
170	Dr Theodore Shennan, 71 Leamington Terrace,	1897
	Dr J. Sibbald, 18 Great King Street,	1859
	Professor Simpson, 52 Queen Street,	1859
	Dr A. A. Scot Skirving, 29 Drummond Place,	1897
	Dr A. T. Sloan, 22 Forth Street,	1885
175	Dr Andrew Smart, 15 Rutland Square,	1865
	Dr G. D. Smith, 148 Ferry Road,	1877
	Dr James Smith, 1 Parson's Green Terrace,	1891
	Dr John Smith, 11 Wemyss Place,	1856
	Dr J. Cormack Smith, 9 Brunton Place,	1896
180	Dr William Smith, 14 Hartington Gardens,	1890
	Dr John Stevens, 2 Shandon Street,	1892
	Professor Sir T. Grainger Stewart, 19 Charlotte Square,	1861
	Dr J. S. Stewart, 15 Merchiston Place,	1885
	Dr Robert Stewart, 42 George Square,	1892
185	Dr William Stewart, 146 Ferry Road, Leith,	1889
	Dr H. J. Stiles, 5 Castle Terrace,	1889
	Dr S. Stirling, 4 Coates Crescent,	1891
	Professor R. Stockman, University of Glasgow,	1891
	Dr A. Stodart-Walker, 30 Walker Street,	1894
190	Dr John Strachan, Dollar,	1867
	Dr C. A. Sturrock, Dunfermline,	1894
	Dr Allan C. Sym, 144 Morningside Road,	1889
	Dr William G. Sym, 20 Alva Street,	1889
	Dr W. Taylor, 12 Melville Street,	1871
195	Dr C. C. Teacher, 16 Newington Road,	1887
	Dr C. H. Thatcher, 8 Melville Crescent,	1876
	Dr R. Thin, 38 Albany Street,	1890
	Dr Alexander Thom, Viewfield, Crieff,	1884
	Dr Alexis Thomson, 32 Rutland Square,	1887
200	Dr John Thomson, 14 Coates Crescent,	1887
	Dr T. J. Thyne, 2 Dean Terrace,	1898
	Dr John Tod, 93 Ferry Road, Leith,	1895
	Sir John Batty Tuke, 20 Charlotte Square, *President*,	1864
	Dr F. J. Turnbull, 6 Randolph Place,	1896
205	Dr Dawson Turner, 37 George Square,	1890
	Dr Logan Turner, 20 Coates Crescent,	1892
	Dr Underhill, 8 Coates Crescent,	1872
	Dr Norman Walker, 7 Manor Place,	1891
	Dr D. Wallace, 11 Rutland Street,	1887
210	Dr Douglas C. Watson, 22 Coates Crescent	1894
	Dr P. H. Watson, 16 Charlotte Square,	1856
	Dr A. D. Webster, Belleville Lodge, Blacket Avenue,	1883
	Dr George R. Wilson, Mavisbank, Polton,	1892
	Dr J. Lockhart Wilson, Duns,	1888
215	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
	Dr T. D. Wilson, 10 Newington Road,	1880
	Dr Russell E. Wood, 9 Darnaway Street,	1883
	Dr W. Fraser Wright, Bonnington Mount, Bonnington Ter.,	1894
	Dr John Wyllie, 44 Charlotte Square,	1868
220	Dr P. A. Young, 25 Manor Place,	1870
	Dr R. J. Erskine Young, 8 Alva Street,	1892

(*b.*) Members Exempted under Rule V. from paying the Annual Subscription.

		Date of Admission.
	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
	Dr D. H. Anderson, 14 *Hartington Street, Barrow-in-Furness*,	1887
	Dr Archibald, 2 *The Avenue, Beckenham, Kent*,	1882
225	Dr J. A. Armitage, 28 *Waterloo Road South, Wolverhampton*,	1887
	Dr W. Badger, *Penicuik*,	1882
	Dr Edwin Baily, *Oban*,	1883
	Dr Andrew Balfour, *Portobello*,	1874
	Dr Andrew Balfour,	1895
230	Dr James Craig Balfour, 17 *Walker Street*,	1884
	Dr J. H. Balfour, *Portobello*,	1881
	Dr Alexander Ballantyne, *Dalkeith*,	1872
	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
	Dr George T. Beatson, 7 *Woodside Crescent, Glasgow*,	1895
235	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*,	1889
	Dr Benjamin D. C. Bell, *Kirkwall*,	1889
	Dr G. J. H. Bell, *Bengal Army*,	1884
	Dr G. H. Bentley, *Kirkliston*,	1877
	Dr J. S. Beveridge,	1861
240	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*,	1865
	Dr P. G. Borrowman, *Elie, Fife*,	1893
	Dr D. G. Braidwood, *Halkirk, Caithness*,	1889
	Dr David Brodie, 12 *Patten Road, Wandsworth Common, London, S.W.*,	1865
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*,	1884
245	Dr John Brown, 68 *Bank Parade, Burnley, Lancashire*,	1878
	Dr J. Macdonald Brown, 48 *Mildmay Park, London, N.*,	1883
	Dr R. D. Clarkson, *Falkirk*,	1896
	Professor Cleland, *The University, Glasgow*,	1864
	Dr Coghill, *Ventnor, Isle of Wight*,	1870
250	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
	Dr John Connel, *Peebles*,	1876
	Dr William Craig, *Cowdenbeath, Fife*,	1894
	Dr J. R. Crease, 2 *Ogle Terrace, South Shields*,	1885
	Dr J. Frank Crombie, *North Berwick*,	1897
255	Dr P. M. Deas, *Wonford House, Exeter*,	1868
	Dr Archibald Dickson, *Hartree House, Biggar*,	1871
	Dr A. Halliday Douglas, *The Neuk, Peebles*,	1842
	Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*,	1889
	Dr C. E. Douglas, *Cupar-Fife*,	1894
260	Dr J. J. Douglas, 42 *Centrall Hill, Upper Norwood, London*,	1891
*	Dr W. B. Dow, *Dunfermline*,	1879
	Dr J. W. Eastwood, *Dinsdale Park, Darlington*,	1871
	Dr F. A. Elkins, *The Asylum, Sunderland*,	1893
	Sir R. B. Finlay, Q.C., M.P., *Middle Temple, London*,	1864
265	Dr Simson C. Fowler, *Juniper Green*,	1892
	Dr F. W. Foxcroft, 33 *Paradise Street, Birmingham*,	1893
	Dr Dyce Fraser, *Chili*,	1883
	Dr J. Hosack Fraser, *Bellfield, Bridge of Allan*,	1895
	Dr R. Freeland, *Broxburn*,	1879
270	Professor Gamgee, 8 *Avenue de la Garve, Lausanne, Switzerland*,	1863

		Date of Admission.
	Dr William Gayton, *Bartram Lodge, Fleet Road, Hampstead, London, N.W.*,	1886
	Dr F. M. Graham, *Willowbridge, Market Drayton*,	1894
	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr George K. Grimmer, *South Queensferry*,	1897
275	Dr Groesbeck, *Cincinnati*,	1875
	Dr W. B. T. Gubbin, *Redland, Bristol*,	1893
	His Excellency Dr R. H. Gunning, 12 *Addison Crescent, Kensington, London, W.*,	1846
	Dr John Haddon, *Honeyburn, Hawick*,	1883
	Dr Archibald Hall, *Montreal*,	1853
280	Professor D. J. Hamilton, *The University, Aberdeen*,	1876
	Dr J. W. Hamp, *Penn Road, Wolverhampton*,	1887
	Dr A. W. Hare, 10 *Cleveland Parade, Darlington*,	1883
	Dr J. Home-Hay, *Alloa*,	1880
	Professor J. Berry Haycraft, 1 *St Andrew's Place, Cardiff*,	1889
285	Dr Stanley Haynes, *Malvern, Worcestershire*,	1864
	Dr R. Dundas Helm, 3 *Alfred Street N., Portland Square, Carlisle*,	1892
	Dr R. E. Horsley, *Jud-Falls, Stoneyhurst, Lancashire*,	1886
	Professor A. W. Hughes, *King's College, London*,	1889
	Dr James Hunter, *St Catherine's, Linlithgow*,	1890
290	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*,	1887
	Dr J. Carlyle Johnstone, *The Asylum, Melrose*,	1882
	Dr D. W. Johnston, *P.O. Box* 2022, *Johannesburg, South Africa*,	1893
	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*,	1871
	Dr J. Keay, *District Asylum, Inverness*,	1887
295	Dr A. J. Keiller,	1889
	Dr George Keith, *Moidart Cottage, Currie*,	1845
	Dr Skene Keith, 42 *Charles Street, Berkeley Square, London, W.*,	1885
	Dr W. Scott Lang,	1886
	Dr Herbert Littlejohn, *Scarborough*,	1892
300	Dr W. H. Lowe, *Woodcote Lodge, Inner Park, Wimbledon, Surrey*,	1845
	Dr Robert Lucas, *Dalkeith*,	1875
	Dr F. R. Macdonald, *Inveraray*,	1860
	Dr K. N. Macdonald, 21 *Clarendon Crescent*,	1880
	Dr W. B. Macdonald, *Dunbar*,	1888
305	Dr John A. Macdougall, *Cannes, France*,	1875
	Professor J. M'Fadyean, 101 *Great Russell Street, London, W.C.*,	1888
	Dr John Mackay, *Aberfeldy*,	1881
	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*,	1893
	Professor M'Kendrick, *The University, Glasgow*,	1870
310	Dr James Mackenzie, 66 *Bank Parade, Burnley, Lancashire*,	1894
	Dr W. O. M'Kenzie, D.I.G.H., 37 *Belsize Park Gardens, Hampstead, London, N.W.*,	1845
	Dr T. J. Maclagan, 9 *Cadogan Place, Belgrave Square, London, S.W.*,	1875
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle*,	1882
	Dr John Macpherson, *Stirling District Asylum, Larbert*,	1891
315	Dr S. Macvie, *Chirnside*,	1897
	Dr J. W. Martin, *Burnfoot, Cluden, Dumfries*,	1888
	Dr A. Matthew, *Corstorphine*,	1882
	Dr E. A. Mills-Roberts, *Bryn Meurig, Bethesda, Bangor*,	1897
	Dr J. Moolman, *Cape of Good Hope*,	1877
320	Dr E. C. Moore, 2 *Coates Place*,	1892
	Dr A. E. Morison, *Brougham Terrace, Hartlepool*,	1889

		Date of Admission.
	Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne*,	1882
	Dr D. G. Macleod Munro, *Alloa*, ...	1898
	Dr J. Ivor Murray, *Granby House, St Nicholas Cliff, Scarboro'*, ...	1857
325	Dr Andrew Scott Myrtle, *Harrogate*, ...	1859
	Dr Leith Napier, *The General Hospital, Adelaide, South Australia*, ...	1879
	Dr T. Goodall Nasmyth, *Cupar-Fife*, . . .	1884
	Dr Ernest F. Neve, *Srinagar, Kashmir, India*, . .	1884
	Dr T. Wyld Pairman, *H.M. Prison, Lyttelton, New Zealand*,	1884
330	Professor Bell Pettigrew, *St Andrews*, . . .	1873
	Dr Alexander Peyer, *Zürich*, ...	1893
	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-Sur-Mer, France*,	1878
	Professor W. S. Playfair, 38 *Grosvenor Street, London, W.*, .	1857
	Sir W. O. Priestley, M.P., 17 *Hertford Street, Mayfair,*	
335	*London, W.*, ...	1854
	Dr J. H. Pringle, 172 *Bath Street, Glasgow*, . . .	1886
	Dr S. Hale Puckle, *Bishop's Castle*, ...	1885
	Dr G. M. Robertson, *The Asylum, Murthly, Perthshire*, .	1890
	Dr J. Maxwell Ross, *Avenel, Maxwelltown, Dumfries*, .	1882
	Dr J. R. Home Ross, *Burmah*, ...	1888
340	Dr Joseph C. Ross, *Withington*, ...	1884
	Dr S. Rumboll, *Hope Villa, Hillary Place, Leeds*, . .	1887
	Dr Thomas Russell, 27A *Westmuir Street, Parkhead, Glasgow*, ...	1888
	Dr Gordon Sanders, *Cannes, France*, . . .	1892
	Dr F. A. Saunders, *Grahamstown, South Africa*, . .	1884
345	Dr Thomas R. Scott, *Musselburgh*, ...	1884
	Dr C. A. E. Sheaf, *Ovalu, Fiji*, ...	1871
	Dr G. F. Shiels, 229 *George Street, San Francisco*, . .	1887
	Dr W. H. Shirreff, *Port Fairy, Victoria, Australia*, . .	1883
	Dr William Simmers, *Denburn, Crail*, . . .	1894
350	Dr T. Skinner, 6 *York Place, Portman Square, London, W.*,	1856
	Dr John Smith, *Brycehall, Kirkcaldy*, . . .	1889
	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia*, ...	1892
	Dr Van Someren, *Goldhurst Terrace, South Hampstead, London, N.W.*, ...	1845
	Dr Somerville, *Galashiels*, ...	1877
355	Dr A. M. Stalker, 140 *Nethergate, Dundee*, . . .	1893
	Dr Graham Steell, 96 *Moseley Street, Manchester*, . .	1877
	Professor Stephenson, *University, Aberdeen*, . . .	1861
	Dr Robert Stirling, 4 *Atholl Place, Perth*, . . .	1891
	Dr H. R. Storer, *Newport, Rhode Island, U.S.*, . .	1855
360	Dr John Struthers, *Ngamakwe, Transkei, South Africa*, .	1895
	Dr J. F. Sturrock, *Arima, Broughty Ferry*, . . .	1887
	Professor J. Symington, *Queen's College, Belfast*, . .	1878
	Professor Lawson Tait, LL.D., 195 *Newhall Street, Birmingham*, ...	1870
	Dr C. Templeman, *Sanitary Office, Bell Street, Dundee*, .	1891
365	Dr Thin, 63 *Harley Street, Cavendish Square, London, W.*, .	1861
	Dr J. Stitt Thomson, *The Mount, Lincoln*, . . .	1877
	Dr J. Batty Tuke, jr., *Balgreen, Murrayfield*, . .	1886
	Dr R. S. Turner, *Keith*, ...	1867
	Dr T. Edgar Underhill, *Dunedin, Burnt Green, Worcestershire*, ...	1885
370	Dr A. R. Urquhart, *Murray House, Perth*, . . .	1893
	Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj, Bombay, India*, ...	1895
	Dr A. B. Whitton, *Aberchirder*, ...	1886
	Dr Oswald G. Wood, *Indian Army*, ...	1886

		Date of Admission.
375	Dr G. Sims Woodhead, 1 *Nightingale Lane, Balham, London, S.W.*, ...	1883
	Dr Yellowlees, *Gartnavel Asylum, Glasgow*, . . .	1862
	Professor John Young, *The University, Glasgow*, . .	1860

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

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(4.) *Diseases of the Skin.*

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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXVII., 1897-98

Meeting I.—November 3, 1897

Dr Argyll Robertson, *President, in the Chair*

I. Election of Members

W. A. M'Cutchan, L.R.C.P. and S. Ed., Hereford, and E. A. Mills-Roberts, M.B., C.M., Penrhyn, were elected Ordinary Members of the Society.

II. Election of Office-Bearers

The following gentlemen were elected office-bearers:—*President*, Dr J. Batty Tuke; *Vice-Presidents*, Dr James Carmichael, Dr Francis Cadell, and Dr P. A. Young; *Council*, Drs Joseph Bell, J. W. B. Hodsdon, R. Milne Murray, D. Noël Paton, Argyll Robertson, Michael Dewar, John Thomson, and J. Shaw M'Laren; *Treasurer*, Dr M'Kenzie Johnston; *Secretaries*, Dr Graham Brown and Mr J. M. Cotterill; *Editor of Transactions*, Dr William Craig.

III. Exhibition of Patients

1. *Prof. Annandale* exhibited a man forty-two years of age, from whom he had removed six weeks previously the entire

larynx, portion of the trachea, and a large vascular tumour of the thyroid gland. He was first seen by Dr Cullen of St Boswells and Dr Rutherfurd of Kelso, and then by Dr M'Bride, who, on examining his larynx, found a malignant growth in its interior. He was admitted into the Professor's ward in the infirmary for operation, and as the breathing was becoming much obstructed owing to the spreading of the interlaryngeal growth, tracheotomy was performed low down. This operation was difficult owing to the enlargement of the thyroid gland; and when the trachea was reached, it was three inches from the surface. Two weeks after, the operation for the removal of the larynx was performed. A new and improved apparatus was shown which had been successfully used in the administration of chloroform, and the temporary ligature of the trachea immediately above the tracheotomy wound was advocated as it had been employed in this case, and prevented all blood or other matters from passing into the air passages during the operation. After the larynx had been removed, the trachea was stitched to the margins of the wound in the neck. The œsophagus was treated in the same way, an indiarubber tube being inserted and kept in position by a silk stitch. For a time the patient was fed through the tube inserted into the œsophagus, but at the end of three weeks the œsophageal tube was removed and feeding carried on through a tube passed by the mouth down the œsophagus. Now the wound was closed except that opening into the trachea, and the patient was able to take and swallow by the mouth, mince, porridge, and other nourishment. In order to prevent undue contraction of the new portion of canal the patient kept in the œsophagus an indiarubber tube, the upper end of which he kept concealed in the mouth with a thread attached, by means of which he drew out the tube when he required to swallow food. There was also shown an improved permanent tracheotomy tube which had its upper end curved downwards, and a piece of indiarubber tubing connected to it so as to lessen the risk of matters passing into the air passages, and also to lessen the risk of cold air affecting these organs.

The parts removed at the operation were also shown.

2. *Mr A. G. Miller*, exhibited a case of RECURRENT FIBROID TUMOURS OF THE FOREARM, after resection of a

portion of the ulnar nerve with restoration of function after grafting.

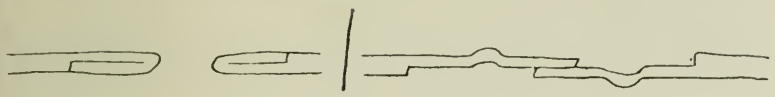
D. F., æt. 73, labourer, Kirkcaldy, admitted to Ward 12, 7th June 1897, complaining of pain in the right hand and forearm.

History.—Patient has been operated on nine times during the last nine years for recurrent tumours in the right forearm.

Present condition.—There is constant pain in the ulnar side of the right hand shooting up to the elbow. The pain is worse at night and is affected by weather changes. The right arm is smaller and colder than the left. There are several cicatrices on the posterior and inner aspect of the forearm, some of them adherent to the ulna. Several hard nodules can be felt under the skin on the inner side of the olecranon.

The tumours removed at the former operations were found to be recurrent fibroids (Paget's). They were growing from the deep fascia of the forearm and periosteum.

Operation, 14th June.—Three fibroid growths, evidently of the same nature as formerly, were freely removed. They were growing from the intermuscular septa of the forearm. One, about the size of half a walnut, completely surrounded the ulnar nerve where it passed between the two heads of the flexor carpi ulnaris. The nerve was dissected out of the tumour, but as it was considerably thickened, flattened, and adherent to the tumour it was considered advisable to remove the involved portion (about 1½ in.). It was found impossible to bring the divided ends of the nerve together. They were therefore split, portions turned up and down, and the ends stitched together in the way recommended by Létiévant. The method is best demonstrated by a diagram, thus—



The evening after the operation sensation was absent in the little finger and half the ring-finger. Next day, however, there was some sensation, and by the 21st June (a week after the operation) sensation and motion were almost perfect. After this the function of the nerve gradually and almost entirely disappeared. There was extension of the terminal phalanges,

glazing of the skin, and atrophy of the muscles, especially in the interosseous spaces. By the third week there was distinct improvement, and, the wound being quite healed, the patient was sent to the Convalescent. After three weeks there he came back with the function of the nerve almost completely restored. He said that he felt the hand as good as ever. He was therefore allowed to go home and to work.

The wound healed by the first intention.

Remarks.

1. The condition of the nerve at the part involved in the tumour resembled neuritis. Thus we have an explanation of the pain.

2. The almost immediate restoration of sensation is very remarkable, suggesting many interesting questions which would be difficult to answer. For instance, the portions of nerve turned down and up must have had their blood supply cut off, and were practically dead tissue. Then these portions must have carried nerve impulses the opposite way to which they were accustomed. Again they were united laterally and not end to end. Once more, some three inches or more of the newly-made nerve was only half a nerve at best.

3. As to the tumours removed. These seemed to be exactly the same as those formerly removed. Paget speaks of each recurring tumour being softer and more malignant-looking than the former. This man has been operated on ten times, and the last tumours removed seemed much the same as the first.

3. *Dr Allan Jamieson* exhibited—

(*a*) TWO CASES OF LUPUS VULGARIS treated with Koch's new tuberculin.

A. K., 17, Edinburgh. Admitted to Ward 38 Royal Infirmary, 3rd August 1897. She had suffered from the disease as long as she can remember, but it had improved under remedies applied during the last twelvemonths by Dr Norman Walker, as an out-patient. The parts affected are the neck, extending pretty well all round, and in front to the level of the clavicle, over the right side of the face, particularly involving the ear; there are also some patches on the left side. There are isolated patches of small dimensions above the left eyebrow, and in front of the left axilla. When admitted there

was so much thickening and infiltration of the tissues that she could not turn her neck with any ease. There was considerable crusting but no ulceration. Scraping was resorted to by Dr Walker, and subsequently some patches were painted with liq. arsenicalis. Since the 1st September, when Dr Jamieson took over the ward, the local treatment has in the main consisted in the application of a 2% solution of potass. permanganatis in water, under oiled silk or guttapercha, for two or three days at a time, then when this had rendered the parts tender, this was intermitted till the irritation had subsided, and again resumed. This was varied by painting with liq. antimonii chloridi. The injection of tuberculin was commenced on the 4th August, and has been pretty regularly continued every second day till now. The initial dose was one-tenth of a milligramme, and this has been gradually raised to three and a half milligrammes, at which it has remained since the 10th of October. On the whole the reaction has been slight or nil, but on one occasion the temperature reached 103·5°, and several times 102° and 101°. The skin over all the affected parts has become soft and pliable, the nodules though superficial are still quite evident, but the interstitial inflammation has disappeared.

Mrs S., 40, Musselburgh. Admitted 23rd August 1897. The disease here also is of long standing, affects the entire forehead, the right cheek extending into the concha of the ear, and part of the left cheek. The skin is more deeply implicated than in the former case. The skin was much reddened, nodulated and thickened. The local treatment has consisted mainly in the application of liq. antimonii chloridi, sometimes alternated with the 2% solution of potass. permanganatis. Her skin is, however, more tender than A. K.'s, as shown by a tendency to erysipelas, and its readily swelling up when the permanganate was applied. The initial dose of tuberculin was half a milligramme, and this caused next day a rise of temperature to 101°, and the appearance of a red rash on the chest and back, closely resembling that of scarlet fever, so that she was sent for three days to the Observation Ward. The reaction in her case has been a more marked phenomenon, the temperature reaching on one occasion 102·6°, several times 100° or 101°, generally exhibiting some rise. Though the dose has been gradually raised, it has been found that about one milligramme is as much as she can stand. There has been no extension of the

disease since she came under treatment, and it has very considerably flattened down.

The conclusion one may draw from these cases is that the combined treatment has produced a greater improvement than would probably have been effected by local measures alone. At the same time there is nothing approaching a cure in either, and it is to be feared that once removed from the warm atmosphere of the ward, the care, the cleanliness, and the rest, the disease will again progress.

(*b*) A CASE OF LUPUS ERYTHEMATOSUS.

H. M., 35, labourer, Edinburgh. A man of only fair intelligence, and of rather intemperate habits. The disease began five years since on the malar portion of the right cheek, spread across the nose to the left, and untreated, it slowly grew worse till about eighteen months ago, when the whole face except the forehead had become affected ; he applied at the Royal Infirmary, and was admitted into Ward 37, where he remained a month, and left suddenly, considerably better. A fortnight since it began to swell up and he was re-admitted on the 27th October 1897. The entire face, with exception of the forehead, and including both ears, and extending on to the nape of the neck, is involved, but the scalp is not yet attacked. The surface is reddened, the hue in many parts, but especially behind the ears, being more of a bluish purple tint. This fades momentarily on pressure in some places, in others does not. In addition to the usual features of lupus erythematosus, viz., erythematous redness, tending to assume a marginate arrangement, with slight scaling and scarring here and there, it presents others. Thus there is a little of the telangiectic type visible, at the alae of the nose and on some outlying patches on the neck. It also exhibits a deep thickening of the skin resembling elephantiasis. Thus the ears are greatly enlarged and stand out straight from the head. The skin behind the ears and downwards over the jaw is much thickened, irregular and uneven, and indeed all the affected part presents more or less irregularity of surface. Though certainly an instance of lupus erythematosus, yet it approximates to those elephantoid forms seen, chiefly on the extremities, in cases of lupus vulgaris.

(*c*) A CASE OF DERMATITIS HERPETIFORMIS.

J. R., 59, mason, from neighbourhood of Edinburgh. Has

suffered from the complaint for ten years. Treatment has always caused a temporary improvement, but it has invariably recurred. The arms were shown covered with the erythemato-bullous eruption.

IV.—Exhibition of Specimens

1. *Dr Argyll Robertson* exhibited under microscopes, four specimens of a rare form of parasite in this country—the Filaria loa. One interesting point in connection with these specimens was the circumstance that they were all removed from one patient, there being no case hitherto reported in which more than two worms had been removed from the same patient. The case was further of much interest as the parasites had been extracted from different parts of the body; one from under the conjunctiva, one from the substance of the lower eyelid, one from under the skin in the left lumbar region, and the fourth from under the skin of the right mamma. Hitherto it was supposed that this parasite was only to be met with under the conjunctiva or in the eyelids, but it was now evident that it might appear under the skin of any part of the body.

2. *Prof. Annandale* exhibited—

Specimens from an interesting case of laparotomy. The special interest was that three different kinds of tumours were removed at the same time.

(1) A large Fibroid of the uterus.

(2) A large cystic tumour of the left ovary.

(3) A dermoid cyst, which contained hair, of the right ovary.

The patient, a woman 48 years of age, was now convalescent.

3. *Mr C. W. Cathcart* exhibited a SUPPURATING HYDATID CYST simulating left sacro-iliac disease.

Helen L., æt 19, was admitted to Ward 5 on 21st October 1897, complaining of a painful swelling over the back of the left iliac crest. It had recently given way and was discharging a sero-purulent fluid.

Five weeks before she had been seized with considerable pain on the left side, shooting from the knee to the shoulder, but worst about the lower ribs, especially when she tried to draw a deep breath. After a fortnight the swelling appeared

in the region of the left posterior superior iliac spine, and as she limped in walking a diagnosis of sacro-iliac disease was made, and she was sent into the Infirmary.

The swelling was red, and brawny looking, and tender to the touch, but she had no pain when the iliac crests were pressed together.

The swelling was considered to be a subacute abscess of uncertain origin. It was more severe and rapid in onset and showed more definite signs of inflammation than is usual with a cold abscess. It was, however, treated as such by opening and scraping. Some gelatinous looking films removed along with the discharge suggested the possibility of a hydatid cyst, but nothing definite was then made out. A few days afterwards several more gelatinous looking films and a considerable amount of pus came away when the patient was being dressed. The pus contained the characteristic hooklets of hydatids. The patient had only come from Shetland a few months before she was taken ill. She had worked at a farm there where several collie dogs had the run of the kitchen.

V. Original Communications

1. VALEDICTORY ADDRESS

By D. Argyll Robertson, M.D., LL.D., F.R.C.S.Ed., President

It now devolves upon me, gentlemen, in accordance with inexorable custom, to deliver a few valedictory remarks ere quitting the presidential chair to which you were kind enough to elect me two years ago. Were I to consult my own feelings I should confine myself simply to an expression of sincere thanks for the great honour I have, by your courtesy, enjoyed, and my obligations to you all for the kindly consideration I have invariably experienced in the discharge of my official duties. But such an infringement of the ordinary course of procedure would be apt to be misconstrued and considered evidence of a want of appreciation of the distinction you have conferred on me, and I have therefore decided to follow the usual custom and review very shortly the work that has been done here during my period of presidency, and refer to other matters affecting the prosperity and efficiency of our Society.

I may remark at the outset, that I have been particularly

struck with the excellent attendance at the meetings and the deep interest taken in the subjects brought forward. My connection with the Society now dates back thirty-six years, and as for several years I held office as one of the secretaries, I am in a position to testify that the life and activity manifested now is in no way less than it was in years gone by. I remember well when secretary that there often was a difficulty in obtaining material sufficient for the various meetings of the session, but the question now generally is how to find time for the efficient consideration of the important matters brought forward. In fact, the sittings seemed to me to be if anything rather unduly prolonged so as to get through the business on each of the billets. This unfailing supply of communications, many of them of much importance, is, I think, to be ascribed in part to the enlargement of the medical and surgical staff of the Royal Infirmary and Sick Children's Hospital, whereby all available material is more thoroughly utilised, in part to the increased number of lecturers in the Medical School of the Royal Colleges, but also to a great extent to the opportunities for original investigation afforded by the various laboratories of the University and by the laboratory of the Royal College of Physicians. Not only we, the members of this Society, but the medical profession generally, owe a deep debt of gratitude to the Royal College for their enlightened policy in instituting such an excellently equipped laboratory in our midst, and for the handsome and generous way in which it is managed. It has supplied a felt want—a want which formerly obliged many earnest, industrious workers to proceed to continental schools to pursue courses of research for which no opportunities or appliances existed here. Now such inquiries can be prosecuted with thorough efficiency at home, and the discussion of part, at any rate, of the results of such inquiries adds greatly to the interest of our meetings. The Royal College of Surgeons is following in the wake of the sister college, and is instituting a separate laboratory of its own, from which also we may expect valuable contributions in the future.

In reviewing generally the work of the last two sessions I may say that the papers included admirably detailed reports of a number of cases of great medical or surgical interest, some of them cases of injury or disease of very rare occurrence, and all illustrating points of diagnosis or treatment of very great prac-

tical importance. Many of the best discussions of the sessions were elicited in connection with such reports. As examples of this I might perhaps instance Dr Gibson's paper on the results of surgical measures in a series of cerebral cases, a subject that appealed about equally to physicians and surgeons; Dr Beatson's original paper on the treatment of inoperable cases of carcinoma of the mamma; and Dr Elder's paper on Agraphia.

In the domain of therapeutics we had also instructive contributions, in which the actions and use of strophanthus, the Nauheim and Schotts method of treating cardiac affections, and the constitution and use of infants' foods were subjected to careful discriminative analysis. I have already incidentally referred to the valuable contributions in which the results of researches in the laboratory of the Royal College of Physicians were brought under the notice of the Society. These were by no means the least important of the papers read at our meetings, and contained useful additions to our knowledge of the mode of excretion of oxalic acid from the body, and the action of the liver upon the fatty elements of foods.

As relating to the general health and well-being of the community, we had an excellent statistical paper by Dr Gillespie, in which the influence of the weather on the occurrence of influenza was illustrated by the number and character of the cases admitted to the Royal Infirmary at various periods of the year. We had also Dr W. G. Sym's paper, in which he detailed the precautionary measures that might be adopted by the State to prevent the occurrence of ophthalmia neonatorum; and perhaps in this category we might also place Dr James' most interesting and philosophical contribution on the treatment of crime, which came as a refreshing variety to the ordinary class of papers read at our meetings.

One important matter associated with measures for guarding the public health, namely, the construction of the New City Hospital for the reception and treatment of infectious diseases, was brought under the notice of the Society through the instrumentality of Sir Henry D. Littlejohn, our most energetic and able officer of health. The proposed plans of the institution were fully explained and the opinion of the Society solicited.

And it is satisfactory to record that a general approval of the plans was expressed.

As far as my recollection goes, this is the only occasion on which the subject of hospital construction has been in any way brought before the Society since the time when what was termed the "battle of the sites" was fought in the hall of the Society. At that time there was a division of opinion in both lay and medical circles as to whether the new Royal Infirmary, which it had been determined to build, should be erected on the site of the old institution, or built on the extensive open ground in which George Watson's Hospital was then situated. The matter was fully thrashed out in debate at more than one meeting of the Society, and I think I am warranted in saying that the result of the discussion (which was the expression by a large majority of the opinion that the new site was the better one) was the turning point in the controversy, and led to the erection of that noble pile of buildings that faces the Meadows. This was an important precedent, which was attended with the best consequences, and I venture to express the hope that when any important change in or addition to the Royal Infirmary, the Sick Children's Hospital, or the City Hospital is contemplated, the plans may be brought for discussion before this Society. Thereby the interest of the profession in these institutions will be strengthened, some useful hints may be obtained, and the ordinary proceedings here be occasionally varied by considerations of hospital construction. Somewhat bearing on this point I have grounds for hoping that the novel electrical appliances that are being introduced into the Royal Infirmary will form the subject of a communication next session.

I must not omit to mention the admirable lantern demonstrations illustrative of pathological structures we were favoured with during the past sessions—nothing could be more beautiful than some of the preparations thus clearly exhibited to all.

To Dr Dawson Turner we were indebted for the exhibition of a series of photographs taken by means of the Röntgen rays, which served well to illustrate the uses to which this new addition to our diagnostic instruments might be put, while Mr Stiles exhibited some beautiful skiagraphs of a hand and forearm in which the arteries had been injected with mercury. I daresay you have all inspected the wonderful dissections of the arteries of the foot and hand to be seen in our museums,

and marvelled at the infinite patience and neatness of hand that must have characterised the dissectors. What a labour and, I may add, what a waste of time, except for the manipulative skill which may have been thus somewhat developed! As far as accuracy, minuteness of detail, and instructive value are concerned, the skiagraphs exhibited by Mr Stiles easily surpass these dissections.

We may, I think, express the confident hope that when the methods of employing the Röntgen rays in medicine become more perfect any important deviation in form or structure of internal organs will be readily demonstrated and the course of disease and the effect of remedies upon it studied with an exactitude to which we hitherto have been unable to attain. In particular I should anticipate the possibility of diagnosing at a very early stage atheromatous degeneration in the aorta and other large internal arteries. It struck me, too, that in such a case as that of Dr Bramwell's, of calcareous degeneration of the heart and arteries in a young man, photographs taken by the Röntgen rays would have exhibited the condition admirably. Unfortunately the man was in so enfeebled a state when he applied for relief that it would have been difficult or impossible to have obtained them.

One important part of the work of the Society consists in the exhibition of patients presenting some characters of interest, and in this department the last two sessions were particularly rich—physicians and surgeons and specialists all contributing freely. If I may be permitted in particular to specify any classes of cases, I should refer to the many admirable examples of the excellent results obtained from operative interference in abdominal and cerebral affections, cases in which exactitude of diagnosis on the part of the physician and skilful operation on the part of the surgeon were alike worthy of admiration. These cases illustrate well the immense advance that has taken place in our knowledge and methods of treatment compared with what existed in the early period of my professional career.

The pathological specimens exhibited, too, were of the most varied character, and illustrative of almost all kinds of diseased states. In fact, if all the preparations shown during the last two sessions were collected together they would form an excellent nucleus of a pathological museum.

A variety of instruments of interest, because of their novelty

of construction or of their practical utility, were brought under our notice, and added their share to the value of our meetings.

I have thus very shortly touched on the subjects discussed at our meetings. Were I to deal with the communications according to their merits, this evening would be far too short for the purpose, and wherein would be the benefit? I would simply weary you. Are not all the papers and other communications admirably reproduced in the elegant volume so ably edited by our energetic member Dr Craig, to whom the Society is deeply indebted for his faithful and assiduous labours? I may therefore, I think, turn to other matters on which I desire to make some remarks, bearing on the work and efficiency of our Society.

I am glad to be able to congratulate the Society on its material prosperity. During the last two years the number of our ordinary members has risen from 360 to 371, and if to this number we add our honorary and our corresponding members, our total membership amounts to 417. We have to lament the loss by death of four of our corresponding and nine of our ordinary members. By the death of Professor du Bois Reymond the name of one of the most distinguished corresponding members is removed from our list. To know him was to respect and admire him, not merely for his admirable method of working, or for the advance he had accomplished in our knowledge, particularly of the physiology of nerve and muscle, by his ably-conceived and accurate researches, but also for his kindly disposition, his unaffected simplicity of character, and his friendliness and hospitality to those who came from a distance to study in his laboratory.

With regard to the loss we have sustained by the death of Sir Thomas Spencer Wells, little need be said. He was universally recognised as one of the foremost gynaecologists of his day, and it was mainly to his indomitable courage and acumen (at a time when many of the highest surgical authorities condemned it in the strongest terms), that we owe the recognition of ovariotomy as a justifiable operation.

The other two corresponding members whose deaths we have to deplore are Sir John Eric Erichsen and Professor C. J. Ask of Lund—the former a surgeon of the highest standing, universally respected, both as a gentleman and a skilful surgeon, as well as one whose surgical writings were received with the highest

appreciation everywhere; the latter, a much respected Swedish surgeon who devoted his attention mainly to gynaecology.

Death has robbed us of nine ordinary members. It is only within the last few days that we heard with regret of the sudden death of Dr F. W. Barry, who was one of our ordinary members. As Senior Medical Inspector of the Local Government Board he had distinguished himself as a most able and valued adviser in all sanitary matters. His reports on cholera in 1892 and 1893, and on epidemics of typhoid fever and small-pox in different parts of England, evinced his sound judgment and thorough acquaintance with sanitary science. The suddenness of his death while on an official tour of inspection came as a shock to all his friends.

Of the others, four did not reside in Edinburgh or the neighbourhood, and the remaining four did not take any very active part in the proceedings of the Society. Two gentlemen have resigned their membership, and we have elected one honorary, one corresponding, and twenty-two ordinary members.

It is a source of satisfaction to me to observe the very flourishing condition of the Society, which, though it may be said to have reached mature years—having been instituted in 1821—still retains all the vigour and activity of youth. At the same time, I may be permitted to offer one or two practical suggestions with the hope of still further increasing the efficiency and attractiveness of our meetings. I offer them for the careful consideration of our future office-bearers, who, I am sure, will only adopt them if they meet with their approval.

It must, I think, have been evident to all that the clinical afternoon meetings have proved a great success. Indeed, the chief difficulty was to get through the amount of business on the billet in reasonable time, so many cases and specimens were offered for exhibition. Now I think this circumstance points to one alteration that might be made with advantage. I think we might have more afternoon meetings in the course of the session. It has often been remarked that comparatively few of our senior members are conspicuous by the regularity of their attendance. But this is not to be wondered at. As age advances the attractions of the fireside in the evening after a day's work, and a soothing dinner, become more and more irresistible, with the result that on the evening of our meetings,

unless the billet contains some paper on a topic specially interesting to him, our senior member resigns himself complacently to the comforts of home. I fully anticipate that afternoon meetings would prove more attractive to this class of our members, while to most of us the afternoon hour would be at any rate as convenient as the evening. Other scientific societies have appreciated this circumstance by holding some of their meetings in the afternoon in place of the evening. If this view prove correct, I do not see why some of our ordinary meetings with papers should not be held in the afternoon also. It may be argued that many of our members may not be able to get through their day's work by four o'clock, but there is nothing against making the hour of meeting later, say five o'clock, which would still enable the business of the meeting to be got through before the ordinary modern dinner hour.

But I have further an alteration to suggest in the procedure at the clinical meetings. You must all have noticed how awkward and inconvenient it is to have patients exhibited during the formal meeting of the Society—the one patient has usually to go the round of the members for inspection before the next one is exhibited, and thus a great deal of time is wasted, and as a rule the inspection is not very satisfactory. Now I would suggest that the patients should all be assembled and exhibited by the physician or surgeon for half an hour before the formal meeting commences, for which purpose not merely the meeting hall, but the side rooms, might be utilised, while each patient should be provided with a very short note of the nature of his ailment. At the end of the half hour the patients should be removed, the President take the chair, and the formal meeting commence. The various cases that have been exhibited may then be mentioned in turn and a very short discussion, limited to three or five minutes, permitted on any case of special interest. After which the business is proceeded with as usual. If this alteration be found to work well, it might be adopted at the evening meetings also. In making this recommendation I am guided by the procedure at the meetings of the Ophthalmological Society in London, at which the course I have described is pursued with the best results.

I am not quite sure whether suitable arrangements could be made to supply members, attending the afternoon meetings

at any rate, with tea. But if this could be done, I think the little expense thus incurred would be amply justified. It would save the time and trouble of going home for the accustomed mild afternoon stimulant on the part of many who might attend the meetings.

Will you permit me one other suggestion? Many present to-night can testify to the great success attending the annual clinical meeting of two or three branches of the British Medical Association at the Royal Infirmary. Now, I see no reason why we might not hold one of our clinical meetings each session at that institution. There are many cases there of the greatest interest and importance which cannot be brought here to be demonstrated at our meetings, but which could thus be inspected, and also there would thus be an opportunity of seeing a number of medical or surgical appliances in use, the employment and value of which could not be so readily gathered from a simple description or exhibition.

In like manner it is worthy of consideration whether a similar meeting—say every second or third year—should not be held in the Sick Children's Hospital. Of course it would be necessary to get the sanction of the Board of Management and of the Staff of these institutions; but there can be little doubt from the success that has attended the application of the British Medical Association Branches that leave would be granted to us.

Perhaps the alterations I have suggested are too many to be carried out at once; but when circumstances are favourable, individual items might be adopted.

It is purely because I am deeply interested in the welfare of the Medico-Chirurgical Society that I venture to submit these proposals. I consider this Society a precious heritage left to our care, one we are bound to foster and cherish to the utmost of our ability. Its aims are of the noblest—to advance the knowledge of our profession, to improve our weapons for meeting and defeating disease, to diffuse a spirit of scientific research, and to fit us more and more for the efficient discharge of our professional duties. It has in the past nobly discharged these functions, and it has proved itself worthy of the task entrusted to it. At its meetings much of the best work accomplished in the medical school of Edinburgh has been brought to the notice of the profession, and most, I

might say almost all, of the leading men in medicine and surgery in Edinburgh, men who have shed lustre on our beloved town during the last three-quarters of a century, have been among our office-bearers, and interested themselves in the work of the Society. I look with confidence to the future, and am convinced that the Society will in no way fall from the high position it has held and continues to hold among the medical institutions of this city.

2. THE ÆTIOLOGY AND TREATMENT OF GLANDULAR ENLARGEMENTS IN THE NECK

By A. G. Miller, M.D., F.R.C.S.Ed., Consulting Surgeon to the Edinburgh Royal Infirmary, and formerly Lecturer on Clinical Surgery, Edinburgh School of Medicine of the Royal Colleges

It is a marvel to me that in these days of investigation and discovery of organisms, and other causes of disease, the very common, evident, and (in many instances) easily removed causes of glandular enlargements in the neck are so often ignored, or at anyrate overlooked. It is the object of this paper, therefore, to point out the importance of discovering, and, if possible, removing the primary causes of gland enlargement. During my fifteen years' experience as Surgeon to the Edinburgh Royal Infirmary, I have seen many cases in which the removal of a bad tooth, or the curing of a chronic pharyngitis, or tonsillitis, would have prevented gland trouble, and saved much disfigurement. *Causa sublata, tollitur effectus*, is an old saying, and I hope to apply and illustrate it in the following argument.

There are many varieties of glandular enlargements that one meets with. I shall, however, refer to only two, viz., the *simple chronic* and the *tubercular*. I have selected these because they are the most common, because they are often difficult to diagnose and differentiate from one another, and because they best illustrate the subject of my paper. Before dealing specially with the *ætiology* of gland enlargements I shall say a few words on *diagnosis*.

Simple enlargements are not usually discovered nor advice sought on account of them until they have existed for some time. The patient is usually a child; often delicate looking; but sometimes quite healthy otherwise. On examina-

tion one finds one or more lumps of an almond shape, firm (but not very hard), not sensitive, freely movable, enlarging very slowly, and with a history perhaps of an acute or subacute attack of inflammation. Sometimes the invasion is insidious.

Tubercular disease of glands on the other hand has three distinct stages. In the first stage the condition is similar to what I have just described. In the second stage softening takes place (caseation) of one or more of the glands when they become matted together. In the third stage there is further softening (liquefaction), and fluctuation can be made out. The skin then becomes adherent, discoloured, and thin. The abscess, so-called, points, and, if not opened, discharges its contents on the cutaneous surface.

Tubercular disease is readily recognised in the second and third stages, but in the earliest stage it is not easily diagnosed from simple non-tubercular enlargement. Indeed, it is sometimes impossible to differentiate the two; because any simple chronic adenitis may become tubercular, and a tubercular affection may commence as a simple adenitis.

If this be true it brings out a fact of great practical importance, viz., that there is a *pretubercular stage.* Personally I am firmly convinced that there is a pretubercular condition in many cases; and I am sure that a very large proportion of the cases of tubercular gland disease that I have seen, might have been prevented by the timely discovery and removal of a carious tooth or some other cause of gland irritation.

It will make things easier if we now consider the anatomical relations of the cervical glands to the various parts of the head, face, and neck from which they derive their lymphatics.

I have found it convenient to divide these glands into the following groups which differ slightly from the groups given in anatomical works. (1) Those at the back of the neck—the *occipital* group. (2) Those behind the ear—the *mastoid* group. (3) Those in front of the ear—the *parotid* group—which may be again divided into the *superficial* and the *deep.* (4) Those under the jaw—the *submaxillary* group. (5) Those lying along the sterno-mastoid muscle—the *sterno-mastoid* group—which may also be conveniently divided into the *superior* and *inferior* (6) Those above the clavicle—the *supra-clavicular* group.

These various groups of glands receive their lymphatics as

follows (so far as I have been able to make out):—The occipital from the posterior part of the scalp; the mastoid from the scalp and ear; the parotid from the front of the head, the ear, and several other parts. This group, however, requires further differentiation, and therefore I have divided it into the superficial and deep parotid groups. The former glands are served by vessels from the front of the scalp, the external ear and meatus; while the latter are connected with the orbit, nose, pharynx, middle ear and upper teeth. The submaxillary glands derive their lymph from the cheeks, lips, mouth and lower teeth. Of the sterno-mastoid groups, which, along with the submaxillary glands, are those most frequently affected, the upper series is connected with the tonsils, pharynx, œsophagus and larynx; while the lower ones are related to the deeper structures, and are generally found to become affected secondarily to the upper ones. The last group (supra-clavicular) is connected with the intra-thoracic and axillary glands, and derives its lymph from thoracic regions mostly.[1]

The clinical importance of this grouping will be evident if we take some examples.

A child has its occipital glands enlarged or suppurating. Employing the above classification as our guide, we at once examine the back of the head, and will most likely find some eruption or pediculi, or both, as the cause. If the posterior auricular glands (mastoid) should be affected, we examine the scalp and external ear. If the submaxillary glands be inflamed, we look at the lips and into the mouth for the source of irritation. If the glands in the upper cervical region be enlarged, there is a much wider range of investigation opened up. We may find the cause in the nose, throat, or middle ear. Once more, if the glands in the lower cervical region be affected, we will probably find, after investigation of the other groups of glands, that the upper cervical, or mediastinal, or axillary glands have been diseased first, and that the irritation has spread by contact to those lower cervical and supra-clavicular groups. I have noticed tubercular disease of these last-named glands associated with old-standing, quiescent, or perhaps to all appearance cured, phthisis.

Having demonstrated the importance and clinical bearing

[1] The superficial (subcutaneous) glands are connected with the skin, and are affected by cutaneous irritations such as boils, etc.

of the anatomical arrangement and relations of the cervical glands, we are in a better position for discussing the *ætiology* of glandular affections.

Causes, in my student days, were usually classified into predisposing, exciting and maintaining. In the present day they are apt to be disposed of under one term, viz., *organisms*. Without desiring to undervalue in any way the influence of germs, I shall adopt the old-fashioned method at present as being more convenient.

1. *Predisposing causes* of glandular enlargement in the neck certainly exist. I shall refer to two, viz., childhood and constitution or heredity. No one can deny that children suffer from gland affections more than older persons, and, whatever explanation may be given, or definition offered, of the terms "constitutional proclivity" and "diathesis," there can be no reasonable doubt that some persons are more liable than others to gland affections, and also that this tendency seems to "run in families."

2. *Exciting causes* might almost be classed under the one head *organisms*. But we cannot get rid of the matter thus easily, for the question naturally arises, How do the organisms obtain access to the lymphatic glands? In answering this question, we have to consider two points—first, the organisms (or their products) have to be absorbed by the lymphatic vessels; and second, there must be a favourable surface from which absorption can take place. These conditions are usually supplied by broken skin or mucous membrane, an inflamed surface, or a lodgment, in some "pocket" or cavity, of septic decomposing matter. I have already described how such open doors are provided—a scalp eruption, a carious tooth, adenoids, tonsillitis, pharyngitis, middle ear disease, and so forth. These various pathological conditions provide the organisms, their products, and the surface capable of absorbing mischief. The natural function of the lymphatic glands is to receive the absorbed materials, to filter them, and to make them innocuous. When this is accomplished all is well and all ends well, but sometimes the septic poison is too strong, or the glands fail in properly performing their function, and then we have glandular infection, irritation and inflammation. We may say, then, that the exciting causes are organisms and their products, plus those conditions of mucous membrane, teeth,

etc., which favour their lodgment and absorption by the lymphatics.

3. I now come to the third class of causes, and to this class I wish to draw special attention. The *maintaining cause* of glandular affections is often the persistence of the exciting cause as I have described and defined it. Remember that I am speaking of simple chronic and tubercular gland affections in the neck. These would not exist at all were there not some maintaining cause. A temporary inflammatory enlargement often appears and disappears along with the primary cause. If the infection be intense or the glands weak, the irritation may lead to suppuration—the gland is destroyed, suppurates out, and there may be an end of the matter. If, however, a gland once irritated remains enlarged, refuses, or is unable to recover, there must be some explanation, some cause of this. This cause I call the maintaining cause, and the maintaining cause, is not unfrequently the exciting cause unremoved. Let me press the argument yet further. Seeing that the organisms and their products cannot obtain access to the glands without an open door such as I have described, I take it that the real maintaining cause is the open door. Let me illustrate this. The glands of the neck become enlarged in connection with a tonsillitis or pharyngitis. If these conditions be cured, the glands will recover of themselves. Again, the glands under the jaw are enlarged and painful in connection with a bad tooth. Let the tooth be extracted and the glands will cease from troubling. The irritation and enlargement of the glands disappear when the cause and source of the irritation are removed. But if the irritant be not removed the irritation is continued, the glandular enlargement persists, or gets worse, and the patient suffers from chronic glandular enlargement.

Now this raises two points which must be dealt with. (1) In some cases glandular enlargements (chronic adenitis) remain after all apparent cause has disappeared. (2) Some cases persist after the cause is removed, and the open door properly shut. Examples of these conditions (which are practically the same) are common in the experience of all, I am sure. In the former the tonsillitis or pharyngitis has had time to disappear, but the glandular irritation (or its effects) remains. In the latter case the tooth is extracted or the adenoids removed, but the glandular enlargement persists.

What is the explanation? It is one and the same in both cases, viz., some irritating factor has lodged in the gland itself, which the gland cannot destroy or expel, possibly on account of its natural inability to do so. That factor, I take it, is an organism, usually the tubercle bacillus. It has entered by the open door, and refuses to be expelled—it has come to stay.

This brings up another interesting point. *Persistent glandular enlargement after removal or disappearance of all discoverable sources of irritation may mean that the glands have become tubercular.* This, if true, is a most important fact. I have become convinced of it both by the course of reasoning which I have just detailed, and also by my experience. Let me put it this way. Is there doubt and difficulty in deciding as to whether, in a given case of glandular enlargement in the neck, the condition is simple or tubercular? If, on careful investigation, no evident source of irritation be found, or if some irritating cause has been discovered and removed, and yet the glands do not improve, then the presumption is that the glands are tubercular, or are at least in the pretubercular stage to which I have referred previously.

Going back then to *maintaining causes* I would narrow them down for practical clinical purposes to two, the *open door* and the *tubercle bacillus.*

This brings me to my "practical application," in other words, to treatment. What I have to say will be very brief. (1) In all cases of glandular enlargement in the neck look for the cause and treat it, or have it removed at once, and generally the glands will get well of themselves, or with the help of some simple local application such as a carbolic compress.[1]

Many patients have been sent to me for excision of glands, whom I have been able to send home in a few days well or nearly so, after the extraction of a stump or removal of some such source of irritation.

No local treatment is appropriate or of any avail so long as there is an irritating cause present. Remove the cause and the glandular enlargement will disappear.

Causa sublata tollitur effectus.

(2) Secondly, if all visible cause has been removed, or has disappeared, and yet the glandular enlargement remains, *remove*

[1] I have already drawn attention to the efficacy of carbolic lotion applications in reducing simple inflammatory affections. *Vide The Hospital,* 1st and 8th July 1893.

the glands, even though you may not be quite certain that they have actually become tubercular. So long as there is chronic glandular enlargement there is risk of tubercular infection. My experience has been that, when glands have been removed "on suspicion," they have almost invariably turned out to be tubercular. I do not think it is safe to wait till one is perfectly certain. By that time, in all probability, more glands will have become infected, and the operation of excision will, when performed, be more severe, while the possibility of extirpating the whole disease will have become more doubtful.

I would sum up the argument of my paper in the following aphorisms :—

1. Glandular enlargement has always a *cause* which should be *sought for* and *removed* if possible.

2. If the cause be not removed the enlargement will *persist*, and such *persistence* (the open door) may give occasion to *tuberculosis.*

3. Persistent enlargement, after removal of all discoverable causes, generally means tubercular infection or a *pretubercular* condition ; therefore all persistently enlarged glands should be excised.

Meeting II.—December 1, 1897

DR J. BATTY TUKE, *President, in the Chair*

The President on taking the Chair thanked the Members for his election.

I. ELECTION OF MEMBERS

The following gentlemen were elected Ordinary Members of the Society :—Dr David James Graham, Dr L. Macvie, Chirnside, and Dr R. J. Johnston.

II. EXHIBITION OF PATIENTS

1. *Dr J. W. Martin* exhibited a CASE OF APPARENT BASEDOW'S DISEASE in a woman 36 years of age, that had been treated with Splenic and Thymus Ext. Tabloids con-

secutively, with benefit. The symptoms, which were of an aggravated kind, lasted over six months, and were characterised by severe vomiting, palpitation, abdominal pulsation, slight protusion of the eyes and enlargement of the thyroid gland. The vomiting, which became severe every five weeks, was combated only with minute doses of calomel and the drinking of potash water. It consisted of the ejection of large quantities of green and yellow bilious fluid, accompanied with much mucus discharge. The sucking of ice and the administration of belladonna somewhat benefited the abdominal pulsation, but as a rule medicines could not be tolerated. There was marked emaciation and cachexia at certain stages of her illness, and at times of severe vomiting required to be fed by nutrient Enemata. There were certain trophic changes in the hair coming out in patches and becoming altered in character like tow. There was suppression of menstruation from the time of first noticing the swelling in the neck. The patient at present complains of dyspnœa on exertion, but otherwise she has no untoward symptoms, and the swelling of the thyroid is not so marked. She is steadily improving in health and gaining weight, though still taking the thymus gland tabloids.

2. *Dr Allan Jamieson* exhibited several cases illustrative of his paper read at this meeting.

III. Exhibition of Specimens

1. *Mr Alexis Thomson* exhibited—

(*a*) Hydatid cysts removed from the left lobe of the liver of a woman, aged 30; the specimen consisted of a quantity of daughter-cysts, estimated at about 1½ pints, which had been extracted from the parent-cyst by means of a dessert-spoon. There was no fluid in the cyst. The latter was not adherent to the abdominal wall, so that the peritoneal cavity on being opened, was shut off by gauze packing, and at the conclusion of the operation, the parietal peritoneum was stretched to the edges of the opening in the cyst; the latter was stuffed with gauze and made to heal from the bottom. The tumour formed by the cyst, projected below the left costal margin and had been observed for two years. There had been a fox-terrier dog in the house for three years.

(*b*) A SEGMENT OF SMALL INTESTINE, PRESENTING A CIRCULAR ZONE OF NECROSIS CAUSED BY THE PRESSURE OF A BAND; the necrosis involved all the coats of the bowel, had given rise to peritonitis, and was on the point of rupture. The specimen was removed from a single woman, aged 53, who, some years previously, had had inflammation of the bowels. The obstruction, for which the operation was performed, had existed for five days. Some bloody mucus had been brought away by an enema. Strangulation by a band like a crow-quill was found in the left iliac fossa; on dividing the band, the proximal constriction recovered; the distal presented the necrosis above described. The damaged portion of bowel was resected and the ends joined together with a Murphy-button. The patient died the same evening.

2. *Dr Harvey Littlejohn* exhibited—

(*a*) RUPTURE OF A TUBAL PREGNANCY.

M. H., æt. 28, who had borne three children, was following her employment in a paper works when she was struck on the back of the head and neck by a hinged board falling upon her. She was stunned, but was able to walk home. Her mother noticed that she was pale, and she complained of pain at the back of the head and across the stomach. She took some supper and went to bed saying her head felt dizzy and light. Next day she stayed at home but got up and went about the house. The next morning she went to her work, but after walking about half a mile, she had to return on account of feeling dizzy. She went to bed and during the day complained of pain in the abdomen, especially upon the right side, while it was noticed that her right hand was swollen and she had no power in it. The following morning she took a cup of tea at 8.30, but afterwards became unconscious, and died at 10.30.

Post-mortem.—Body well nourished, face and lips very pale, slight *post-mortem* lividity on underlying portions of body. Abdomen appeared distended. Breasts contained milk.

On opening the abdomen it was found to contain a quantity of clotted blood which filled an ordinary hand basin. The left side of the uterus near the fundus was enlarged to the size of an apple, and on the upper and anterior aspect of this tumour there was a rent one and half inches long from which the

enruptured chorion protruded. The left ovary and fallopian tube was firmly bound down by adhesions. The right ovary contained a Corpus Luteum the size of a threepenny piece.

The fœtus was $2\frac{5}{8}$ inches long, and the pregnancy between the ninth and twelfth week. There were no external marks of injury on the back of the head or neck. There can be little doubt that the rupture occurred from a sudden strain or shock at the time of the accident, but it is interesting to note how comparatively slight the hæmorrhage must have been at first, and that severe symptoms only came on on the third day after the accident when she attempted to proceed to her work.

Of interest from the legal point of view is the question of the liability of the employer under such circumstances, presuming that it could be shown that the accident had resulted from a cause for which he was responsible. The direct injury was trivial, but indirectly it caused death, though it may fairly be said that the condition present would in all likelihood have produced a fatal result within a very few weeks.

(*b*) THREE SPECIMENS OF RUPTURE OF AORTIC ANEURISM. In all the cases the men were following their employments at the time when the rupture occurred, and were unaware that they were suffering from the condition.

The first case was that of man, æt. 52, who had had some shortness of breath and spitting of blood for two years. The aorta was very atheromatous and about 2 inches above the valves, on the posterior aspect, there was a round opening the size of a halfpenny, which led into a cavity lined with firm laminated clot, the size of an orange. The left bronchus was compressed, and in its mucous membrane there were two openings communicating with the aneurism. The upper, the size of a pin head, and the lower running transversely, and $\frac{1}{3}$ of an inch in length. The upper opening had probably existed for a considerable time and accounted for the constant spitting of blood.

The second case was that of a man, æt. 62, who had been complaining of shortness of breath for some time, and who suddenly fell down while walking in the street.

The ascending portion and arch of the aorta was dilated to twice its normal size, and internally was covered with calcareous plates, varying in size from a halfpenny to a sixpence. Immediately above the valves was a rent 2 inches in length through

the two inner coats. The effused blood had dissected between the coats down as far as the abdominal aorta, and caused a large extravasation in the region of the left kidney.

The third case occurred in a bootmaker, æt. 46, who, while engaged at his work, suddenly uttered a cry and sank down. He had complained of pain in his side for some years together with shortness of breath.

The ascending aorta had an opening the size of a five shilling piece on its left side which led into an aneurism extending into the left pleural cavity, and which had ruptured into the lower part of the pleural sac.

3. *Mr J. M. Cotterill* exhibited—

(*a*) A FIBROUS STRICTURE FROM THE LARGE INTESTINE. The patient, male, aged 24, had suffered for four years from symptoms resembling those of relapsing appendicitis. When admitted into Mr Cotterill's wards a few weeks ago, he had been suffering from a severe attack, which had come on a few days before. He had severe pain in the right iliac fossa, vomiting and constipation.

A small hard tumour could be felt through the abdominal wall, situated just below the edge of the lower ribs on the right side, and immediately below this mass there appeared, when the attacks of tenesmus came on, a tumour of about the size of a cocoanut, resonant, evidently a distended and hypertrophied portion of gut.

Mr Cotterill exposed this tumour by a 5-inch incision; found it to be, as he expected, a fibrous tumour of the large intestine; removed it together with about 5 inches of adherent and hypertrophied gut; and joined the small intestine, which was divided 2 inches or so above the ileo-cæcal valve to the ascending colon, by means of a bone bobbin.

The patient made a very good recovery.

(*b*) A PERFORATED VERMIFORM APPENDIX. The patient, a boy, aged 14, was admitted with symptoms of general peritonitis, and in a condition of collapse. Mr Cotterill exposed the appendix, and finding it gangrenous and perforated, removed it in the usual way, leaving a short stump of it attached to the cæcum of about $\frac{3}{4}$ of an inch in length. The abdomen was very carefully washed out. The interest of the case consists in the fact that the patient recovered from the shock and peri-

tonitis well, and was going on apparently to a rapid recovery, when on the sixth day after operation, symptoms of perforation of the gut reappeared, attended by severe peritonitis. Mr Cotterill again exposed the part, and found that the stump of the appendix, which had been left, and which had to all appearance been perfectly healthy, had sloughed off flush with the surface of the large intestine. The edges of the perforation were therefore turned in and stitched by two rows of fine silk. The patient again recovered well from the operation; but a few days later was attacked by peritonitis again, and succumbed. It was found at the *post-mortem* that there were two small circular perforations of the small intestine, situated several inches above the site of operation, and these had caused perforation of the ileum.

IV. Exhibition of Instrument

Dr J. O. Affleck exhibited an adaptation of the Phonendoscope which he had found useful for purposes of clinical teaching. It consisted simply in the removal of the outer disc and the lengthening of the ear tubes. Experiments had been tried with tubes of over twenty feet in length, and cardiac murmurs and pleuritic friction were made out quite plainly at this distance; but tubes of 12 feet were found to be more easily manipulated. The tubes could be passed round a large class of students during a clinique, and, in the case at least of very well-marked physical signs, were able to convey a fair impression to the listener. Quite recently in a case of aortic disease with loud murmurs, where the patient was far too ill to bear any handling, advantage was taken of his being sound asleep to bring the instrument now described into use. The nurse sat by, holding the disc lightly over the precordial region, while the ear-tubes were passed round a class of over fifty students, who all heard the murmurs plainly, the patient being all the while asleep, and there being absolutely no fatigue or discomfort to him by the examination. Of course the physical signs could not by this plan be so clear as they would be on closer examination; nevertheless, inasmuch as it is a desideratum in clinical teaching that as many as possible should have an opportunity of obtaining an impression of morbid sounds in typical

cases, where this can be done without injury to the patient, the method now referred to, or some similar contrivance, would seem to go a certain way towards accomplishing this object.

Dr Affleck demonstrated to the Society the use of the instrument in a case of heart disease, with well marked murmurs.

V. Original Communications

THE SERUM DIAGNOSIS OF ENTERIC FEVER

By CLAUDE B. KER, M.D., C.M.,
Medical Superintendent of the Edinburgh City Hospital

It is now a year since Sheridan Delépine brought the serum test for enteric fever prominently before the profession in this country. Since the publication of his admirable paper in the *Lancet* a large amount of evidence has accumulated in favour of the accuracy of this new method of making a diagnosis, which has always been admitted to be of considerable difficulty. It is not my intention in this paper to recapitulate the history of the discovery and evolution of this test. That has been made familiar to most of us by the various papers which have from time to time appeared in the British medical journals, notably by those of Wright and Semple, Fison, and Delépine. It will be sufficient to remark that the honour of first publishing this discovery undoubtedly rests with Widal of Paris, although Grünbaum, working independently, appears to have reached the same conclusions at least as soon as the French observer. The test itself, as is now well known, depends on the behaviour of the Eberth's bacillus when exposed to the blood serum of an enteric patient. When in such a medium the bacilli lose their motility and agglutinate into clumps, whereas in a normal blood no such reaction occurs.

It is with the routine use of this test in hospital practice that I am at present concerned. The fascinating questions regarding the cause of this reaction I must leave to physiological chemists and bacteriological experts. All that I am able to contribute to the literature of the subject is a very brief summary of the results obtained in the Edinburgh City Hospital during the current year. And these results may be of more interest from the fact that the blood of all these cases

was tested in the hospital by the same individual who was responsible for their diagnosis and treatment. In fact, we test the blood of our enteric patients as we test their urine, as an ordinary piece of hospital routine.

Owing to various circumstances I was unable to start this method of diagnosis in the hospital till the 1st of February. Since that date every case sent in as "enteric" or as "observation enteric" has had the test applied. For the first three months I received great assistance from Dr Donald Hutchinson, who collaborated with me and did fully half the work, though we always checked each other's results. Since April, when he left the hospital, I have done all the tests myself with the exception that during the month of August Dr Forrester, who took my place in the hospital, very kindly continued to make the necessary examinations. To these gentlemen and to the other assistant medical officers I owe my best thanks.

Technique.

If reliable conclusions are to be drawn from the examination of a large number of blood sera, it is obviously necessary that the test should be applied to all of them in exactly the same manner. The method I have employed was suggested to me by Dr Robert Muir, and is slightly modified from that of Grünbaum. After careful cleansing, the thumb of the patient is pricked just above the nail, and the blood is drawn up into an ordinary Zeiss' leucocytometer pipette to the mark just below the bulb. The point of the pipette is then wiped and sterilised beef bouillon sucked up till the bulb is full. This gives a dilution of one part of blood in ten of bouillon. This mixture is then ejected into a piece of glass tubing of small calibre previously bent into a U shape. The U shaped tube is carefully labelled and placed with others in a centrifuge, when the blood corpuscles separate out and fall into the bend of the tube. There is no necessity for the separation to be very complete, as a few blood corpuscles in the clear serum assist greatly in the rapid focussing of the microscope. The culture used must be not more than twenty-four hours old, and none of the cases here reported were examined with a culture of more than eighteen hours. The stock cultures are kept on gelatine or agar at the temperature of the room. The evening before the test is to be applied a culture is made from one of them on a

new agar tube, which is then placed in the incubator. Next day the culture is always plentiful enough to allow a small loopful to be scraped from it. This is stirred into ten drops of sterilised beef bouillon in a watch glass, and it is the resulting emulsion which is used in performing the test; a definite proportion of the diluted serum being mixed with it on a celled slide and examined under an ordinary high power.

Dilution.

The proper dilution of serum to use, however, is a matter of some debate. The serum of normal people and that of patients suffering from diseases other than enteric, may, if insufficiently diluted, give a marked reaction. It is this fact which accounts for the unsatisfactory results obtained by some observers. At the City Hospital our observations are made on the reaction of a somewhat indefinite quantity of active bacilli moving in a medium consisting of one part of serum in thirty of beef bouillon. We obtained this mixture as follows :—A little of the diluted serum is drawn from the U shaped tube by means of a fine glass pipette, manufactured in a Bunsen flame for the occasion, and a very small drop is placed on the celled slide. Two drops of similar size of the bouillon emulsion of bacteria are placed beside it, and the three drops are then mixed together, and the cover glass is put on. These drops can readily be judged by the eye, or, if preferred, platinum loops can be used to obtain them instead of pipettes. It is obvious then, as the serum in the U tube is diluted 1-10, that we have on the slide one part of serum in about thirty of bouillon, and, in addition, we have the bacilli contained in the bouillon from the watch glass. I may say that the results I have obtained by this method have been remarkably consistent, and I venture to believe that, roughly speaking, the same number of bacilli are in the mixture on each occasion the test is done. But, of course, accuracy as to this point is impossible when we only use a platinum loop to estimate the amount taken from the agar culture. If the culture is thick the loop holds more than when it is thin, and the probability of error is enormous. But I see no reason that the method should be less accurate than that of measuring the dilution by taking a loopful of culture grown in bouillon. It would appear equally impossible in that case to estimate the number of bacilli in the

loopful, and one culture would be very likely to contain more than another.

I must confess that it is only since reading the recent paper of Gruber and Durham that the question of the number of bacilli assumed great importance in my mind. These authors believe that certain substances, which they term agglutinins, exist in varying quantities in the blood of the enteric patient, and cause agglutination by some chemical or physical action on the sheath or protoplasm of the bacillus, and are used up themselves in the process. If the bacilli then are too numerous, a certain number should escape agglutination, and thus partial reactions would be accounted for. Very few of our cases showed such partial reactions, so it is probable that we have not used an excess of bacilli, and that our method, rough as it admittedly is, has answered its main purpose.

Time Limit.

The next point to be considered is how long are we to allow for the reaction to take place. Many time limits have been suggested, from the half hour of Grünbaum to the twenty-four hours allowed by Widal. I have always given four hours. I take a note of the condition of the specimen at the time of mounting, about half an hour later, and then examine it finally when it has been mounted four hours. I may say that the specimens are usually frequently examined between times. If the clumps are not absolutely distinct and the field between them clear when the time limit is up, I consider the reaction negative. If it seems partial I wait a few days and examine another specimen of the patient's serum.

I may here add that we find it advantageous to examine seven or eight specimens at a time. The different sera act most efficiently as checks on each other, and if, as sometimes happens, there is anything wrong with the culture it is certain to be detected. If clinical diagnosis has not already made it practically certain that there is at least one serum, derived from a patient suffering from some disease other than enteric, among those to be examined, we are in the habit of examining a normal blood or the blood from a scarlatina patient to act as a check.

Results obtained.

In the early part of the year Dr Hutchinson and myself examined a large number of blood sera from patients in the different wards of the hospital, including cases of scarlatina, measles, diphtheria, and erysipelas. None of these gave the reaction. I will limit myself here, however, to the cases sent into the hospital as enteric fever or for observation for that disease. These cases are 169 in number and may be classified as follows :—

I. CASES WHICH GAVE THE REACTION.

These may be subdivided into the following groups—

(α) Cases pathologically proved to be enteric.
(β) Cases clinically diagnosed enteric.
(γ) Cases probably enteric.
(δ) Cases possibly not enteric.

II. CASES WHICH DID NOT GIVE THE REACTION.

Subdivided into—

(α) Cases pathologically proved not enteric.
(β) Cases clinically diagnosed not to be enteric.
(γ) Cases probably not enteric.
(δ) Cases possibly enteric.

As regards the question of clinical diagnosis, the greatest care has been taken to secure that such diagnosis should be accurate. The great majority of the cases were diagnosed definitely before the test was applied, and as regards the others we have done our best not to be influenced by the presence or absence of the reaction. The chief points to which attention was paid in making a diagnosis of enteric fever were, the history of the patient, the general course of the temperature, the comparative rapidity of the pulse, the general appearance of the patient, the condition of the abdomen, the size of the spleen, and, lastly, the nature of the stools. I need hardly add that the presence of spots in a certain number of cases assisted the diagnosis greatly. Throughout the year I have had the advantage of having the great experience of Dr Wyllie, the consulting physician to the hospital, at my disposal. During his occasional absences Dr Affleck, Dr James, and Dr Graham Brown have all been exceedingly kind in helping me in difficult cases.

I. Cases giving the Reaction, 121.

(α) CASES PATHOLOGICALLY PROVED ENTERIC.

Eight cases on post-mortem examination showed all the characteristic lesions of enteric fever. They all gave the reaction. In two of them the diagnosis during life was rendered more difficult from the fact that they were complicated by acute pneumonia. A third had the much rarer complication of tubercular meningitis.

(β) CASES CLINICALLY DIAGNOSED ENTERIC.

Ninety-nine cases came under this heading, and all of them gave the reaction. Many were complicated, and on admission were by no means obviously enteric, but the subsequent course of these left no doubt as to the diagnosis. Two are interesting as being complicated with other infectious diseases. One of them was admitted to the wards with typical diphtheria in addition to enteric, while the other had both enteric and scarlatina on admission. In both cases the enteric appeared to have been the original disease. We may therefore conclude that the presence of other toxines does not have any effect on the accuracy of the test.

(γ) CASES PROBABLY ENTERIC.

This group consists of cases which had neither marked symptoms on their admission to hospital, nor any temperature to speak of after their admission. Many, however, were notified by practitioners as enteric fever, and had presumably symptoms at home sufficient to justify such a diagnosis. The remainder were notified as "observation."

These cases were thirteen in number. Of them no less than five had been ill for three weeks before coming into hospital. Three of them were definitely notified as enteric, and two of these had distended abdomens and enlarged spleens. I regard it as quite possible that all these five had the fever at home, and were entering on their convalescence when admitted.

Four cases had been a fortnight ill. The history of these was quite compatible with enteric fever, but none of them had more than two days' fever in hospital.

Four had a history of from a week to ten days' illness. One of these had a brother suffering from the fever in the hospital, and the appearance of two of the others was very much in favour of the diagnosis being correct in their case. I see no reason why we should not accept all of them as abortive cases. In none could we get any history of the patient suffering from the disease previously.

I have seen it suggested in a paper on this subject that the presence of the reaction in cases of such short duration should make us change our views on the subject of the course of enteric fever. Personally I have always firmly believed in the occasional abortion of the disease, and we have recently had in hospital four brothers who illustrate admirably the different course it may run in different individuals, even when exposed to the same conditions. One boy had a three weeks' fever and then a relapse, the second a three weeks' fever only, while the third and fourth were ill fourteen days and thirteen days respectively.

(δ) CASES PROBABLY NOT ENTERIC.

One case, which we believe was probably not enteric fever, gave a definite reaction on three occasions. The patient was a girl of fourteen, who was admitted to the hospital with the history of a fortnight's illness at home. Her main symptoms were constipation, sore throat, and abdominal pain. In hospital she was slightly delirious at night and very stupid by day. Her abdomen was flat and her spleen not apparently enlarged. Her skin showed no rash but was discoloured and spotted with petechiæ, which were diagnosed as flea-bites. On the nineteenth day her temperature began to fall, and the chart suggested the termination of a typhus or pneumonia rather than that of an enteric. The admission of her father and sister with undoubted typhus three weeks later inclines me to the belief that she was really suffering from typhus, and that probably she had not been ill quite so long before admission as was stated to us. We must conclude either that she did not necessarily suffer from the same disease as the rest of her family or that she had really had enteric fever at some earlier date, although a careful inquiry failed to find any history of it.

If we assume her to have been typhus, it is somewhat remarkable that Colville and Donnan, who have examined the

sera of 132 cases in the laboratory of Queen's College, Belfast, only report one case, other than enteric, which gave the reaction, and it also was a case of typhus. They give the same explanation as myself, namely, that the patient had probably enteric fever at some earlier date. The sera of other typhus cases examined by them failed to react, and I have had a similar experience at the City Hospital.

II. Cases which did not give the Reaction, 48.

(α) CASES PATHOLOGICALLY PROVED NOT ENTERIC, 3.

Three cases which gave no reaction were subjected to a post-mortem examination. None showed enteric lesions. One of them, who had suffered from abdominal symptoms for some weeks before admission, came into hospital with a concurrent facial erysipelas, to which she succumbed. A large number of stones were found in her gall-bladder, which was full of pus.

The other two cases were typhus.

(β) CASES CLINICALLY DIAGNOSED NOT ENTERIC.

These cases were forty-one in number. About one-third of them were notified as enteric fever, and the remainder were sent in for observation. It must be remembered that a fair proportion of them were sent in by dispensary students, but the large majority were very difficult to diagnose at the time of their admission.

They were finally diagnosed as follows :—

Pneumonias (of which 9 were croupous and 7 were catarrhal)	16
Typhus	4
Influenza	4
Diarrhœa	5
Tuberculosis	2
Tubercular meningitis	2
Tubercular peritonitis	1
Appendicitis	1
Pleurisy with effusion	1
Constipation	2
Gastro-enteritis	2
Undiagnosed	1
	41

(γ) CASES PROBABLY NOT ENTERIC.

I only class one case under this heading. The patient was an Italian woman, who was six days ill only on admission, and had no symptoms in hospital. She only spoke her native language, and I could get no satisfactory history. I am inclined to believe she had not enteric. An interesting feature in her case was that on two separate occasions her blood showed almost complete clumping in six hours with dilutions of 1-20 and 1-30. As she did not give the reaction within the time limit I class her as giving a negative result.

(δ) CASES POSSIBLY ENTERIC.

These were three in number. Not one of them was an undoubted enteric, but the result of the test caused us some surprise.

(i.) A man, aged nineteen, who was admitted with a history of a rigor six days before. He took to his bed almost at once, and had a high temperature. His bowels were constipated, and he was slightly delirious. Dr Kenneth Douglas, who attended him, sent him in for observation, suspecting either typhus or enteric. He appeared to us more like the latter. His spleen was enlarged and his abdomen a little tumid. He had no rash. His temperature fell rapidly, and we regarded him as an abortive case. After failing to obtain Widal's reaction, we sent his blood to Dr Muir, who also reported a negative result. I regard his chart as compatible with that of an abortive enteric, and the fall of his temperature too gradual for that of a typhus. Moreover, he had a sister at home treated as enteric by another doctor.

(ii.) A girl of twenty-three years of age sent in by Dr Proudfoot for observation. She was admitted on the fourteenth day of her disease. Her symptoms were headache and fever, and her bowels were very constipated. Her general appearance on admission suggested enteric, but her abdomen was soft and her spleen normal in size. Her temperature very much resembled that of an enteric case, and became normal on the twenty-fourth day. An addition of fish to her diet caused the slight rise of temperature so often seen in an enteric. Her blood was tested on five different occasions, with negative results.

(iii.) A woman aged forty-eight sent in by Dr Byrom Bramwell for observation. She had been ill for seventeen days, complaining of languor, headache, and pain in limbs and abdomen. On admission her abdomen was flat, and her spleen only slightly enlarged. She was very constipated, and her temperature was comparatively low, seldom reaching 101° F. She is now convalescent after three weeks of fever in the hospital.

If we call these patients enteric, we certainly have a strong case against the accuracy of the test, but it is worth noting that all three were sent in merely for observation, and that, therefore, their own medical men doubted their being enteric. Further, on the symptoms they presented it needs a bold man to assert positively that they were enteric. The test has been so accurate in my other cases that I am inclined to follow it here, though I can offer no suggestion as to what the patients were really suffering from.

Conclusions.

I think I may claim that this series of cases has given very consistent results, and that we may accept Widal's test as the best method for diagnosing enteric fever. Every large number of examinations is bound to present some anomalies, but we have been wonderfully fortunate in having only four dubious results out of a total of 169.

The age of the patient seems to make no difference to the result. My youngest case was one year old, my oldest fifty years. As all my cases were admitted to hospital after, at least, one week's illness, I have no record of a serum giving the reaction before the seventh day.

The method that we have used is, I consider, very well suited for hospital practice. The use of a centrifuge may be an objection, but every well-equipped institution should be provided with one of these machines. In private practice, however, nothing appears to me more suitable than the method of Delépine. The want of an incubator practically prevents the general practitioner at present using the test. When, however, capsules of dead bacilli are placed at his disposal, this means of diagnosis will be within his reach. I have examined some sera with quite satisfactory results, using capsules kindly sent to me by Dr Hutchinson. Widal and

Wright and Semple have proved that such dead bacilli react perfectly well.

To obtain reliable results it seems absolutely necessary to use a sufficient dilution. Dineur of Antwerp has in a recent paper asserted that the dilution of the serum must be 1-25 at least. Even with the rough and ready method we have used I doubt if our dilution has ever been less than that, and by endeavouring to keep it at 1-30 we have probably always been on the safe side. The advantages of diluting the blood when it is taken from the patient are many. In the first place, one drop is sufficient. Secondly, as Pakes has pointed out, the first contact of a pure serum with the bacilli in the process of mixing may cause ambiguous results.

In conclusion, I have to thank Dr Muir very cordially for his kindness in occasionally providing me with reliable cultures, and still more for having encouraged me to undertake a method of diagnosis which has given us such excellent results.

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2. ECZEMA PALMARE AND PLANTARE

By W. Allan Jamieson, M.D., F.R.C.P.Ed., Physician for Diseases of the Skin, Edinburgh Royal Infirmary

There is a remarkably obstinate form of chronic eczema, which attacks the palms, and, though more rarely, the soles sometimes also. This is occasionally only part of a more widespread development of the disease, but is perhaps oftener seen alone. Prior to its appearance, there may be elicited a history of dryness of the palm, with a degree of associated itchiness, but whether this has been remarked or not, the complaint commonly takes its origin in the centre of one palm, though it is generally not long till both are implicated, possibly, however, to an unequal extent. When such cases come for advice, there are hard scaly patches of infiltrated skin, involving more or less of the surface. There is ragged

and uneven scaling, yet not very pronounced, while in the natural lines of flexion, or independent of these, are deep and painful cracks. The hands feel hot, and burn and itch at times, though this latter feature is scarcely so aggressive as in eczema of other regions. From functional reasons, the integument of the palm is less movable than elsewhere, and the infiltration renders it absolutely fixed. Hence, should the case be a severe one, the hand is habitually held in a half-closed position, any attempt at extension giving rise to much suffering by tearing open the existing fissures or producing new ones. In this way the member is all but useless for work. The morbid condition even in its extremest grade is pretty nearly confined to the palm, advancing sometimes along the fingers towards their tips, the pulp remaining, as a rule, immune.

The secretion of sweat is almost wholly suspended, provided the hands are uncovered, but temporary relief can be obtained by artificially correcting this, by soaking in water, or freely rubbing in oily lubricants. No curative effect is thereby procured, as when the hand is allowed to dry, or the oily application is intermitted, there is rather an aggravation. As Bulkley says: "When kept constantly wet, such hands will be quite flexible, and patients even with a greatly aggravated eruption will sometimes be able to do washing, although each time the hands are dried thereafter, they are worse, and the suffering from them may be intense." [1]

The feet may be similarly affected, but on the soles it seldom reaches such proportions. This is because they are more protected from changes of temperature, while from wearing stockings and shoes a degree of moisture is favoured, so that fissures are not so common nor generally so painful. A symptom is observed there which is not so evident on the palms, namely, the existence of a band of congestion beyond the scaly area, fading imperceptibly into the natural tint of sound skin. This is merely the outcrop of the subjacent hyperæmia, veiled from observation by the thickness of the horny layer itself and the opacity of the abnormal scaly accretions.

Though met with in both sexes, this variety of eczema is most commonly encountered in women, and in them about

[1] "Eczema and its Management," 1881, p. 211.

the menopause. "This discloses its influence," as Jacquet puts it, "in the form of congestive attacks, supplementary to the menstrual flows which have disappeared."[1] At the same time, we meet with instances at a period of life much antecedent to that. There are other factors at work in some cases. One of these is a gouty element, often an obscure one, but possibly connected with the cessation of the periodical loss, the tendency to obesity, and the discontinuance of exercise, or at least the disinclination towards it, observed at that epoch of female life. Another, valid in both sexes, is worry or anxiety, the deleterious effect of which on nutrition there is no need to further insist on. Causes of local irritation may exert a determining influence, but these are frequently impossible to trace in women, occasionally more evident in men.

Turning to the pathology of the affection, we find that the dense resistant film of keratin, which forms the protective surface of the palms, may undergo various morbid changes. One of these is simply hypertrophy. The layer takes on an increase in thickness, either in limited areas, or pretty extensively over the palm or sole. This is known as hyperkeratosis, and finds its exemplification in the congenital and often hereditary form of ichthyosis or tylosis, in callosities (those efforts to protect from the injurious friction of manual labour), or on the foot as corns, the reaction against an ill-fitting shoe. Here there is no more than an exaggeration of a natural process. But there are also anomalies in cornification, and to one of these Auspitz first attached the name parakeratosis, a term which has been adopted by Unna, who has defined, amplified, and analysed it, so that it has now come to occupy a definite place in cutaneous terminology. According to him, it is, reduced to its simplest terms, an œdema of the transitional epithelium, a true parenchymatous œdema, a dropsical condition of the epithelial cells themselves. As a consequence, the prickle layer of the affected part becomes broader than normal, and the state of the granular layer in particular indicates the soakage of the epithelium, for the separation of keratohyalin is arrested, or the granules composing it grow extremely fine, while the layer itself is thicker than is customary. The horny layer, too, is moist, thus less brittle, less coherent

[1] "Saint Louis Atlas of Skin Diseases and Syphilitic Affections," 1897, pt. 9.

than it should be, and separates in masses of flakes, not insensibly as scales. There is delay in the process of cornification, which indeed is never fully accomplished, the epithelial cells drying up in place of undergoing a complex series of changes. "Parakeratosis consists, then, only in an abnormally simple cornification, that is in the absence of the many intermediate stages (separation of keratohyalin, breaking up of the nucleus, drying up of the internal protoplasm) which accompany normal cornification. The cell wall becomes cornified or hardened as usual, but the cell contents remain moister and better preserved. Proper cornification is hindered by the abnormal saturation, the parenchymatous œdema, of the epithelial cells."[1] To this there is added epithelial growth —acanthosis and exudation—catarrhal inflammation, in short.

Now this peculiar anomaly of cornification, along with the other phenomena just mentioned, is seen in a very typical form on the palms and soles, and is in one of its manifestations regarded as eczema. Much the same condition occurs, however, under other circumstances, in accordance with a law well formulated by Jacquet. "All parts of the skin, which are anatomically and physiologically specialised, have a tendency to impress common characteristics upon lesions affecting them, and always in a similar determinate manner. This is particularly true of the palms and soles when the horny type is assumed. Thus lesions of widely different nature (psoriasis, eczema, lichen, syphilides, etc.) tend to become uniform one with another, and also with certain affections special to these parts—such as the essential keratodermias, the nature of which is very imperfectly known."

The diagnosis of such cases is not always easy. Though frequently — erroneously — designated psoriasis palmaris, the difficulty does not lie between this dry scaly form of eczema and true psoriasis, for though psoriasis does sometimes attack the palms, and there gives rise to an analogous condition, yet this is so rare as an affection, independent of psoriasis elsewhere, as to be practically out of court. The same may be said in even stronger terms of lichen planus. There does not appear to be a single recorded instance of lichen planus in which the eruption was limited to the palms. The influence

[1] "The Histopathology of the Diseases of the Skin," English edition, 1896, p. 199.

of syphilis, however, must be rigidly excluded. When the palms are attacked in the secondary and symmetrical period, there can scarcely be much perplexity, since there are sure to be some local or constitutional evidences discoverable. In the tertiary period, again, syphilis rarely invades both palms; it starts from separate foci, is apt to assume a crescentic arrangement, and to heal in the centre while it progresses circumferentially. In the intermediate epoch, however, between the second and the fifth year from infection, we may meet with a keratosis of the palms manifesting itself symmetrically. Sometimes, too, there is a compound of eczema and syphilis, extremely puzzling, and refractory in the last degree. This extends as a diffuse redness over the entire palm, often advancing on to the flexor aspect of the wrist, and terminating there with a defined and crescentic margin. There is comparatively no itching, and if there are fissures, these are superficial and few. The colour also is different from that of eczema; it is more a dull coppery tint, due to the heightened blood hue shining, as Unna has shown, through the syphilitic plasmoma with increased translucency, from the absence of collagenous substance. Nor can we obtain much satisfaction from the therapeutic test, as this is apt to be unsatisfactory, especially in the instances where a blending of the two diseases occurs.

Another form of keratosis of the palms which must be excluded is that consequent on the administration of arsenic. The idea that arsenic is not only "good for the skin," but has a special property of rendering the complexion clear and beautiful, has taken firm hold of the popular mind, and is fostered by delusive advertisements. Valuable as arsenic is as a medicine in certain ailments, if judiciously used, it occasions, if taken even in small doses for a length of time, not merely a dinginess, but a positive pigmentation of the skin generally. On the palms and soles it exerts its stimulant action on the sweat glands, giving rise first to hyperhidrosis of these regions, then to the formation of warty corns round the sudoriparous orifices, and finally, by their coalescence, to a diffuse hyperkeratosis, the surface being covered by a dense yellow investment, associated with burning sensations. Attention to this property of arsenic was primarily drawn by Jonathan Hutchinson, and many examples are now on record. Sometimes the dose has been trifling, or the drug has not been continued long; in such, a

special idiosyncrasy must be admitted. In other instances the keratosis apparently began after the exhibition of the medicament had ceased, yet once started the process tended to encroach on new ground. The hyperhidrosis, the origin at the sweat pores, the warty corns, and the burning sensations, should arouse suspicion.

Occasionally the *Primula obconica* may cause a patchy parakeratosis of the hands, lasting for some time, arising from squeezing the leaves. The lesion as I have seen it is superficial, and is not strictly limited to the palms.

Besides these, there are two rare conditions—the symmetrical keratodermia of the extremities described by Besnier,[1] where there are warty thickenings of portions of the palmar and plantar surfaces, surrounded by an erythematous zone, slowly progressive but not painful, and with no subjective symptoms. Also the cases discussed by Brooke,[2] as erythema keratodes, where a thick covering of yellow horny epidermis appeared on the palms and soles, bounded by an erythematous areola, and accompanied by heat and other inflammatory symptoms. In his cases there was pain and tenderness but no cracks. Our acquaintance with these latter is too restricted to permit of any exact generalisation.

The treatment of this form of palmar eczema has hitherto been unsatisfactory and disappointing. No doubt one could give great relief, and sometimes cure, by the use of agents, such as salicylic plasters or salicylic collodion, which occasioned separation of the thickened masses, and the subsequent application of salves containing lead, such as the ung. diachyli or ung. vaselini plumbicum, to promote a more healthy growth. But the procedure was a slow one, and the disease strongly tended to relapse. The peculiar softening effect of emol keleet, when applied as a thick watery magma, covered with an impervious material to prevent drying, led me at first to hope that by its means a cure might be obtained. But though the immediate results were admirable, the condition soon recurred on discontinuance of the emol keleet. We were still seeking for some substance which would so modify the epithelium, when the accretions had been got rid of, and the surface had become smooth, as to restore normal cornification, when Unna visited

[1] "International Atlas," pt. 2.
[2] *Brit. Journ. Dermat.*, London, 1891, pp. 23 and 335.

Edinburgh about eighteen months since, and introduced to notice his oxidised pyrogallol, as a remedy for lupus erythematosus and psoriasis. Pyrogallol, or pyrogallic acid, had proved itself most useful as an elective destructive agent in lupus vulgaris, and valuable in the dispersion of limited patches of psoriasis, particularly of the scalp, but it was too toxic to employ over large areas, and even on small was liable to evoke a troublesome dermatitis, which in the case of lupus erythematosus led to the appearance of fresh foci of disease. It belongs to those substances which possess great affinity for oxygen, and is therefore called a reducing or deoxidising body. Unna attributed their action to this property, and on various occasions instituted researches into the properties of several members of the group.[1] It occurred to him, however, to further investigate how much this action really depended on the reducing function itself, and how much could be attributed to the operation of the agent when its affinity for oxygen had been satisfied.[2] He had some pyrogallol thoroughly oxidised by exposure to air and ammonia vapour. The brownish-black substance so produced, he found to have no toxic properties, and to occasion no such dermatitis as the unoxidised did, while it was equally effective in causing the disappearance of the patches of psoriasis. When using it in psoriasis, I was struck by the smoothness and polish of the skin left after the fading of the patches. It occurred to me that perhaps in this oxidised pyrogallol would be found the remedy for eczema palmare. A trial was at once made of it in all the cases available, and with gratifying success.

The method of procedure adopted has been the following. The hands (and feet) are enveloped in poultices made of starch jelly, with which some boric acid has been incorporated, *not merely sprinkled on*, applied cold between two folds of cotton cloth. Each time the poultices are changed—once in four or six hours—the palms are briskly rubbed with a rough, though soft dry cloth, and thus the soddened, unhealthy epidermis made gradually to peel off. Not too much is attempted at a time, but in course of four or five days to a week the palms

[1] "Ichthyol und Resorcin als repræsentanten der Gruppe reduzirende Heilmittel," 1886. "Chrysarobin u. Pyrogallussaure in der Praxis," *Monatsh. f. prakt. Dermat.*, Hamburg, No. 3, Bd. ii.

[2] "Neue Thatsachen ueber reduzirende Heilmittel," *Deutsche Med.-Ztg.*, Berlin, 1886, No. 84.

will have become smooth, soft, pliable, and of a pinkish hue; cool, extensible, and free from itchiness. The poultices are now laid aside, and an ointment thus compounded rubbed in well but sparingly each day.

℞ Acidi pyrogallici oxydati	.	grs. 5–30.
Lanolini		semi-unciam.
Ol. amygdalæ,		
Aq. destillatæ . . .		ana drachmas duas.
		M.

To this has in some cases been added 10 grs. of salicylic acid. The ointment blackens the parts to which it is applied, but chiefly such as had been the seat of the parakeratosis. This returns for a time to a slight extent, but can be kept under by washing with a resorcin and salicylic soap, made with a superfatted basis, more or less friction being employed according to circumstances. The effect of the oxidised pyrogallic acid ointment in restoring normal keratinisation is quite remarkable, and, so far as my experience yet goes, it is permanent. Further time must elapse ere one can speak quite definitely as to this, but nothing hitherto tried has at all approached it in rapidity and efficiency.

The subjoined cases have been selected from a series treated in this way, as illustrations of the disease and the results of treatment.

The first case is an interesting one, since the patient was long under treatment by other remedies, which, though obtaining a considerable degree of improvement, all failed to cure the disease.

Case I.—Miss G., a lady about middle age, who has suffered from eczema of the hands for many years, and had been seen by me at intervals during more than six. The disease affected both palms, worst in the centres, but advanced along the fingers, encroaching on their sides; she was otherwise robust, though she occasionally was liable to attacks of indigestion, while the bowels tended to be loose, going on to rather sharp diarrhœa. She engaged voluntarily in mission work of a rather exacting kind, in winter abroad, and was usually worse then. Improvement was effected by the application of salicylic acid in plaster form, by washing with Duncan & Flockhart's

resorcin and salicylic acid superfatted soap, by the application of resorcin in lanoline thinned down with almond oil, or later with whale oil. On 24th November 1896, it is noted that the hands exhibit a pretty firm epidermis, though darkened in places by the resorcin, and still a little dry. She was then given the ointment containing the oxidised pyrogallic acid. This was continued till the last time on which she was seen, on the 20th January 1897, when the condition was better than it has ever been. The palms were then quite smooth, with only here and there a trace of brownish scaling. They felt soft and even perspiring.

The duration of the three following cases, before treatment by the method indicated, was just about a year; the rapidity with which all improved as soon as it was commenced was remarkable.

CASE II.—Mrs R., æt. 48, Berwick-on-Tweed. She came to the Royal Infirmary on 30th June 1897, and was seen by Dr Norman Walker, who prescribed the application of a solution of tar in quillaia. This was soon left off, and she resorted to a boric acid ointment, and afterwards to a balsamic cream, on her own responsibility. When first seen by me on the 27th September 1897, there was great thickening of the horny epidermis of both palms. The hard masses were heaped up in dark blackish and yellowish layers all over the surface of the palms, extending over the flexor aspect of the fingers almost to the tips. The back of the hands was healthy, but there was a little dry infiltrated eczema on the flexor surface of the forearms. Her health was in all respects good. She was ordered to apply the boric-starch poultices till the hands were soft and smooth, rubbing off the layers of accumulated epidermis as they became softened, then to employ the oxidised pyrogallic ointment, which in this case contained a little salicylic acid. On the 21st October she again presented herself. She had continued the poultices for four days only, when, the hands being soft and free from scales, she commenced the application of the salve. The hands were now soft, smooth, cool, and merely a little rough at the roots of the fingers, and still slightly blackened by the acid, but freely extensible.

CASE III.—Mrs B., æt. 48, Dolphington, came to Royal Infirmary on 21st September 1897. The disease had lasted a year; it began on the ulnar side of the left palm, and gradu-

ally spread till all the surface was involved, though worst in the centre. The right soon also was attacked. There was great thickening of the epidermis, and she could not extend or flex the hands with any ease or comfort. The nails and tips of the fingers remained free. The same treatment was prescribed. The poultices were in this instance continued for a week, then the ointment resorted to. On the 16th October the palmar surface of the fingers was now completely well, soft, smooth, of normal thickness, and but little stained with the pyrogallic acid. There were here and there some yellowish spots of hyperkeratosis on the inner edge of the palms, at the root of the ring finger, and at the margin of the ball of the thumbs. The skin felt cool, there were no fissures, and the hands could be freely flexed and extended without discomfort. The appearance of the palms was very much that of a person accustomed to do a little manual labour.

CASE IV.—R. C. M., æt. 40, brassfinisher, Edinburgh, was under my care some years ago with an attack of general eczema, from which he recovered. At present (16th October 1897) only the palms are affected. The centre is of a pale pink tint, and is dry, but there are patches of roughened thickened epidermis, firmly attached, scattered over the palmar surface, with fissures in places. The fingers are also involved nearly to their tips. The nails are healthy. The same treatment was prescribed. He poulticed the hands for ten days, then commenced to use the ointment. When seen on the 30th October, the hands were slightly blackened; the epidermis was, however, already fairly smooth, though the ointment had only been employed four days. There were still here and there some persisting thickened areas, especially on the right near the wrist, where there was a tendency to crack in a longitudinal direction. The surface was cool, soft, slightly pinkish, but the hands could be freely extended.

In the fifth case both the hands and feet were attacked, and in the instance of the former the disease was not limited to the palms, though, after treatment had been begun, it soon became restricted to that part. When last seen the cure was more complete that in the three preceding ones.

CASE V.—P. T., æt. 54, Edinburgh, blacksmith. He had naturally rather hard hands, and did not perspire much on palms or soles. The disease began in June 1897 on palms,

as dry, peeling, itchy patches, but it did not continue limited to that part, but, when seen on the 4th September, the whole skin of the hands was affected, great thickening of the horny epidermis on a reddened substratum, obscured to a considerable extent by the parakeratosis. There was a good deal of infiltration. There were fissures occupying the natural folds, and also running transversely to these. The skin of the tips of the fingers was glossy and tense. The hands were very itchy when exposed to the least friction. The soles were almost exclusively affected. The masses of epidermis were dry, flaky-yellow, on a pinkish ground-work formed by the infiltrated and congested corium. The redness, but not the parakeratosis, extended to the ankles and round the sides of the feet. Though cool, the feet were likewise itchy. He had been previously treated with starch poultices, followed by salicylic plasters, and finally tar; and in consequence the feeling of heat, which had been prominent at first, had ceased, so that both palms and soles now felt cool, but further advance in the way of improvement had become arrested. His manner of life was in all respects regular, and his diet on the whole suitable, though there had been a deficiency of green vegetables. The urine was normal. He was directed to poultice in the same way as the other cases, with boric starch jelly, and, when the parts had become smooth, to apply 5 per cent. of oxidised pyrogallic acid in lanoline cold cream. On the 21st September all the swelling of the hands was gone, the palms were smooth, somewhat stained by the acid, but showing no fissures. The feet had in like manner benefited, but were more blackened. The ointment was applied in lessening quantity, and on the 9th November the hands were practically well, the condition of the epidermis restored to natural, and the soles bore the pressure of walking perfectly. As his work is arduous, he has postponed resuming it till next month.

The results of treatment in the last case to be related are not yet so brilliant as in those previously recorded, yet there has been marked improvement. There was no discoverable fault in diet or mode of life, while the general health was apparently as good as could be. The disease in this instance commenced on the soles, not spreading to the hands till a couple of months later, and after a month of treatment the improvement was most evident on the soles, the parts longest

affected. The case is specially cited with the view of anticipating any idea that a panacea for eczema of the palms and soles is being vaunted. No such assertion is advanced, yet it is quite possible that a further employment of the remedy, the excipient being modified a little, may in this instance also prove in the end successful.

CASE VI.—M. G., æt. 39, builder; a healthy, strong man, who came with his ordinary medical attendant to see me from a country town, on the 27th October 1897. The disease began seven months before in the centre of the sole of both feet. At first there was a degree of moisture. A couple of months later some similar spots appeared on the legs, then on the arms, and finally in the middle of each palm. When examined, the soles were seen to be reddened in the central portion, while on the ball of the heel and over the distal ends of the metatarsal bones the colour is yellow. The redness, which was of a deep purplish tint, extended along the sides of the feet to the breadth of an inch, then faded. There was some scaling on the sole, and, on passing the hand over it, it felt in parts as if there were here and there warty corns, which tended to run together. The soles itch at times, particularly when the feet become hot after walking, or in bed. Scattered over the legs and arms were rough, slightly scabby, yellowish-brown patches, apparently made up of aggregations of conical papules. When these formed they for a time oozed slightly. In the centre of each palm there was a patch of hard, dry, peeling eczema, the size of a florin. His feet never were wont to sweat much, nor do they now. When the complaint commenced he wore heavy boots and thick grey knitted socks. The same treatment by boric starch poultices and oxidised pyrogallic ointment, combined with washing with resorcin salicylic soap after the poultices had been discontinued, was prescribed. He was again seen on the 23rd November, and reported that he had continued the use of the poultices for ten days, when the palms and soles were quite smooth, soft, and pliant. He then commenced the pyrogallic ointment, washing daily with the soap. All the patches on the legs and arms had completely smoothed down, and were nothing more than faint brownish stains. Some others had come out on the thighs and flanks, but had likewise flattened. The red margin to the sides of the soles

had disappeared, but the central parts were still rougher than normal, marked with fine lines, but little, if at all, infiltrated. The horny layer of the epidermis on the surface still had a tendency to split and peel. The sclerosed eczema of the centre of the palm had improved less; there were no cracks, but the part was still dry, hard, and with a liability to split. He was directed to wash at night with the resorcin salicylic soap, and subsequently to rub thoroughly well in a salve made up of 15 grs. of oxidised pyrogallic acid in a mixture of lanoline, whale oil, and water. In the morning, before applying it, to rub the parts with a loofah dipped in water. As the soles still felt hot on walking, to wear inside the shoes an inner sole of loofah, the "Vitalité sole," which carries down the perspiration.

Since the onset of eczema palmare is generally insidious, and that of eczema plantare frequently unnoticed at first, measures of prevention are difficult. The risk of recurrence after cure is best obviated by maintaining the healthy state of the skin of those regions. In some cases oatmeal should be substituted for soap, for detergent purposes. If the latter is employed, only the best varieties of the superfatted toilet soaps are admissible in the case of the hands. The epidermis of the soles is to be kept in order by daily friction with a loofah and cold water.

Meeting III.—December 21, 1897

Dr J. Batty Tuke, *President, in the Chair*

I. Exhibition of Patients

1. *Dr C. W. MacGillivray* exhibited a case after excision of part of the transverse colon for malignant tumour, with subsequent reunion of the bowel by means of a Hayes Button.

John Bell, æt. 41, admitted to Ward 29, under Dr Affleck, in June 1894; complaint, painful dyspepsia, vomiting, and emaciation. This condition had commenced some weeks previously, and was now complicated by constipation, flatulence, and distension; a small nodule the size of a hazel nut to be made out in the epigastrium. Under diet and stomatic

medicine with enemata he improved slightly, and left on July 1894. He was readmitted in November 1894, more emaciated, and with marked dyspepsia, great distension and constipation, vomiting severe and frequent, and, for a few days before admission, fæcal. Dr MacGillivray saw him then with Dr Affleck, and recommended large enemata, which relieved him. The nodule was still occasionally to be felt. The motion became regular, and he was dismissed in December much improved. Readmitted for the second time to Ward 29 in March 1895, in a worse state than before; great distension and fæcal vomiting; nodule much larger; diagnosed to be in transverse colon, the proximal part of which was very much distended. An operation was recommended, and he was transferred to Ward 16, the obstruction having first been overcome by means of enemata. On opening into the abdomen above the umbilicus, a large growth of the nature of a malignant adenoma was found in the transverse colon adherent to the liver and stomach, the colon on the right side being enormously thickened and distended. About 6 inches of the colon was removed along with the growth, but as the upper part of the colon was about 4 inches in diameter, with hard thickened walls, while the lower part was atrophied and collapsed, it was thought better, especially as the patient was in a very weak state, to make an artificial anus. The patient made an excellent recovery, and left hospital in the end of July 1895.

He continued well and strong and gained weight, but the artificial anus caused him such inconvenience that he frequently returned, requesting that something more should be done. He was finally readmitted into Ward 16 in June 1897. Prepared for operation, and an eliptical portion of skin, including the artificial anus, excised; the two ends of the colon, which lay in the midst of a mass of matted omentum, were reunited by means of a Hayes Button, and the skin wound sutured, a small drainage tube being introduced down to the junction of the bowel. He made an uninterruptedly good recovery; passed wind the next day, and two days later passed a motion. On the twelfth day he passed the button in five pieces. He was discharged on 17th August 1897, and has since had daily motions, and has enjoyed perfect health.

2. *Dr John Thomson* exhibited a boy, aged 2½ years, with marked TREMOR OF THE LEFT FOREARM.

The child had had measles last June, and had never been quite well since. Seven weeks ago, his mother noticed his left arm shaking for the first time, and since this began he had not been able to walk. Within the last week or two his left leg had seemed a little stiff. The fingers were flexed on the palm, and the arm held with the elbow at about a right angle, and with the forearm semi-pronated. The movements consisted in a rhythmical coarse tremor of the forearm and hand towards and away from the body. They did not take place when the arm was quite at rest, but always began if the child held his arm from the side or tried to do anything with it.

There was tubercular dactylitis of two of the fingers on the left hand. This had been present before the measles.

The knee jerks were exaggerated on both sides, and the left leg seemed a little stiff.

The movements of the arm had increased in extent during the last month. They were regarded as probably due to a tubercular nodule in the motor area of the cortex, and it was proposed to recommend an operation for its removal.

3. *Dr Alexander James* exhibited two cases of IRRITATION OF THE LUMBAR CORD due to bone lesions.

The first was that of a young man, G. S., aged 19, who was admitted to the Royal Infirmary November, 13, 1897, complaining of a pain in his left leg, and difficulty in walking. He told us that he had had a fall on his left knee three years ago, as the result of which a small nodular thickening showed itself in front of the head of the tibia, just below the attachment of the patellar tendon. This little nodule had increased in size, and become tender on pressure, so that kneeling was impossible, and for this he had been discharged from the army two years before. Two and a half months ago he was run down in health, and an eczematous rash made its appearance on both legs. For this and for the difficulty of walking he came to the Infirmary. On admission we found marked impairment in the voluntary motor power in the left leg and thigh muscles. The muscles of the left leg and thigh felt softer than those of the right, and measured one inch less in circumference. Skin sensibility was unaffected, but he com-

plained of an occasional numb feeling in the feet, especially the left. The plantar reflex was markedly increased in the left leg, slightly so in the right. The knee and ankle jerks were also markedly increased in the left leg, and less in the right, and knee and ankle clonus were present, specially well marked on the left side. He also had trouble in micturition, often being able to pass water only once in the twenty-four hours. Dr James had no doubt that the impaired functional and irritable condition of the lumbar cord was due to the tender nodule in front of the head of the tibia, and in this connection he showed again the patient whom he had brought before to the Society in February 1896. In this lad the irritability of the lumbar cord had been due to irritation in connection with the tarsal bones of the foot. The source of the irritation had been found to be tubercular disease, and amputation had been performed at the lower third of the leg. The interesting point was, that since the amputation the irritable condition of the cord had quite disappeared. The only trace of it was a slight increase in the left knee jerk, but this was getting less and less as months went by.

4. *Dr J. O. Affleck* exhibited a patient (a man aged 44) upon whom Loreta's operation for stricture of the pylorus had been performed in November. The case was one of chronic gastric ulcer, symptoms of which had existed for about fifteen years. Latterly these had become so aggravated, and the patient's condition so reduced, that the possibility of malignant disease suggested itself. Chemical analysis of the gastric contents, however, proved the presence in abundance of free hydrochloric acid, and tended to confirm the diagnosis of ulcer.

Treatment by lavage and other means giving him but temporary relief, the patient expressed a strong desire that an operation should be performed in the hope of some means of remedying the condition being found. He was accordingly transferred to the care of Professor Annandale, who has kindly written the subjoined particulars of the operation performed by him.

Operation

The abdomen was opened by a median incision, extending from a little below the xiphoid cartilage to one inch below the umbilicus. When the stomach was exposed it was found

to be much dilated. At a point corresponding to the upper curve, and adjoining anterior wall, and about one inch and a half from the pyloric sphincter, there was felt a hard and flattened mass a little more than one inch by one inch in extent. This mass did not feel so hard as a malignant growth, and was a part of the stomach wall itself. A small opening was now made into the stomach at a short distance from its junction with the duodenum, and pointing in the direction of the hard mass. The finger was then introduced into the stomach through this wound, and passed on into the duodenum, when it was found that the pyloric ring was somewhat contracted, and only admitted the point of the finger. A dilator was introduced, and the ring dilated. It being evident that the thickened wall of the stomach acted as an obstruction, the small incision into the stomach was prolonged through it, thus making the incision into this cavity about two inches in length. This thickened portion when cut through appeared to have a simple fibrous texture,[1] and the inner surface of the stomach corresponding to it was muscular, and had a somewhat soft and granular appearance. The wound in the stomach was now stitched up after Heinchie's method—first, by continuous suture of fine silk through all the coats ; second, by a similar Lambert's suture ; and, lastly, a piece of omentum was brought over the part and secured by a few stitches. Closure of the abdominal wall wound completed the operation. The patient's progress after the operation was all that could be desired, and an excellent recovery resulted.

The patient, as stated, made an excellent recovery from the operation, and was discharged from the Infirmary feeling quite well. He subsequently came several times to report himself, and his appearance testified to a marked improvement in his general health and condition, while the former gastric symptoms had practically disappeared.

NOTE

The sequel to this case is a sad one. On 19th March 1898 the patient was again admitted to Ward 29, this time suffering from an attack of acute bronchitis, following influenza. He stated that up till a week previously he had felt perfectly well, when he was suddenly attacked with influenza, which was accompanied by severe cough and dyspnœa. The patient was evidently in a most serious condition, and despite all remedies which could be employed, sank rapidly, and died on 24th March.

On *post-mortem* examination there was found extensive acute bronchitis of both lungs, with patches of commencing catarrhal pneumonia. The stomach was carefully

[1] A small piece was removed for microscopic examination, and was found to be non-malignant in its nature.

examined, and a large chronic ulcer was found running in an antero-posterior direction across the lesser curvature of the stomach near the pyloric end. It showed very little evidence of healing, but the pyloric stricture, which had previously existed, was entirely gone, and so far the operation performed upon the stomach had been successful.

5. *Mr J. W. B. Hodsdon* exhibited—

(*a*) A boy, aged 3, who had suffered from strangulation of the small intestine, caused by bands in connection with tubercular glands.

After laparotomy and division of the bands the patient made an uninterrupted recovery.

(*b*) A patient whose entire tongue and submaxillary glands had been removed six years previously for recurrent carcinoma.

6. *Dr Stewart Stirling* exhibited a CASE OF LUPUS VULGARIS AFTER NINE MONTHS' TREATMENT.

His object in bringing this case before the notice of the Society is principally to show the results of simple treatment —to a considerable extent hygienic—steadily persevered in for a lengthened period—as compared with the more energetic measures frequently recommended in such cases.

The patient is a little girl, aged 7 years. The disease is of five years' standing. It began on the face during the second year of life. Enlargement of the adjacent lymphatic glands appeared about the same time, so that there is a strong probability of there being some relationship between the two conditions.

When the patient first came under his observation—fully nine months ago—the disease was very extensive. A very large area of lupoid tissue occupied the front and sides of the neck, the under surface of the chin, and a considerable part of the cheeks—especially the left. Smaller sized patches were present on the upper lip, nose, forehead, and right elbow and upper arm. The lymphatic glands in the neighbourhood of the face were enormously enlarged, which added greatly to the disfigurement of the patient's face. The teeth were nearly all decayed and loose, and the gums were swollen and painful.

He commenced the treatment with the syrup of the iodide of iron internally, and by the application of Unna's salicylic acid and creosote plaster to the diseased patches. Meanwhile all the carious teeth were removed. After two weeks of this

treatment he found that the syrup of iron disagreed with the patient's stomach, and that the plaster caused much pain and irritation. Both were consequently discontinued. As a substitute for the plaster he used a modification of Brooke's ointment, chiefly consisting of salicylic acid, oleate of mercury, oxide of zinc and vaseline. This ointment—in conjunction with cod liver oil internally—has been employed up to the present time. Under this treatment the enlarged glands rapidly subsided and healthy cicatricial skin appeared in the diseased areas. The child's health is now excellent, and the disease is almost entirely eradicated. He attributes the highly satisfactory results obtained in this case as much to the liberal use of cod liver oil, as to the local applications.

7. *Mr J. M. Cotterill* exhibited a case of MULTIPLE LIPOMATA. The patient, a female, aged 58, had suffered for some years from several small tumours from about the size of a hazel nut to that of a small walnut, situated, with one exception, on the forearms.

The tumours were small encapsulated fatty tumours, and were peculiar in various ways—(1) their multiplicity; (2) the fact that they were restricted to the forearms; and (3) that some of them were accompanied by very severe pain.

These multiple lipomata are rather rare. Only a few cases have been recorded. They differ in several points from the "painful subcutaneous tubercle," *e.g.*, in position—the latter being most common on the legs, and being chiefly composed of fibrous tissue.

In this patient three of the tumours which were most painful had been removed, and it was found that they were in relation with small cutaneous nerves, but the nerves were not observed to penetrate them, nor to be split up over their surface, but simply lay alongside of the lipomata.

8. *Dr James Carmichael* exhibited (*a*) a boy, aged 2 years, suffering from RICKETY PSEUDO-PARALYSIS. Late walking was common in rachitic children. This was an aggravated case. The signs of rickets in this case were widely open anterior fontanelle; flattened vertex with slight mid-frontal groove; enlarged costochondral joints and enlarged distal radial and tibial epiphysis; delayed dentition;

convulsions and head sweating; muscles very flabby and ligaments lax. The case had been sent into hospital as infantile paralysis, because the muscles of the left leg were atrophical compared with the right leg, the diameter being $\frac{3}{4}$ of an inch less at mid-calf. This was really the interesting point in the case as regards diagnosis. On careful examination of the muscles of the small leg the reflexes, both cutaneous and muscular, were found normal or slightly exaggerated, and the electrical reactions normal, thus showing the absence of any central lesion in the spinal cord.

(*b*) A girl, aged 5 years, with INFANTILE PARALYSIS, affecting the muscles of the right thigh and leg. The case showed all the ordinary signs, very feeble reflexes, reaction of degeneration and muscular atrophy. Under massage daily and weak faradic current applied every other day there was an apparent improvement going on.

(*c*) A girl, aged $2\frac{7}{12}$, who had been three months under observation. She was affected with left HEMIPLEGIA, arm, leg and face being affected. The history was interesting. A year ago she had a convulsion of short duration. Nine months ago she suffered from measles. Two months before admission she had a second general convulsion similar to her first; and two weeks after, or six weeks before admission, she had a severe general convulsion, lasting for sixteen hours. This was not attended with complete loss of consciousness, and for three days after she had twitchings of the left side of body and limbs, followed by total loss of power in her left side and paralysis of the face. The mother states that within a short time there was a slight return of motor power. On admission it was found there was motor paralysis not complete of left arm, leg and face, with spastic rigidity of the muscles, most marked in the arm, the fingers and thumb being rigidly flexed. It will now be seen that the child's recovery is all but complete. In such cases in children the lesion was almost invariably meningeal, and in this case it might be considered probable that, as a result of the long continued convulsions, acute hyperæmia and capillary hæmorrhage had taken place from the pia mater covering the cerebral cortex on the right side. The same result is known to occur during severe paroxysms of pertussis. Convulsions generally precede or accompany this acquired form of hemiplegia, and when of a

less severe nature they may be a symptom due to the same cause as the paralysis itself, thrombosis, embolism, meningitis or pachymeningitis. In this case, the attack being so sudden and the convulsion so severe it seems reasonable to suppose that the hyperæmia resulting from the prolonged fit may have been succeeded by hæmorrhage. In most cases, however, in which recovery takes place, we have no means of determining with certainty whether the fits are concomitant or causal.

II. Exhibition of Specimens

1. *Dr Alexander Bruce* exhibited (*a*) a specimen of hæmorrhage into the middle lobe of the cerebellum, which had subsequently burst into the fourth ventricle. The case was of interest both from its rarity and from the point of view of diagnosis. The symptoms presented some features common to hæmorrhage into the pons and into the lateral ventricles, namely :—coma, contracted pupils, and complete paralysis of the limbs. Both of these sites, however, were excluded by the character of the pulse and the temperature, and by the manner in which the paralytic phenomena appeared. The sudden attack of vertigo was followed first by loss of co-ordination of the lower limbs, and, after a distinct interval, by vomiting and loss of power of articulation, and then by complete paralysis of upper and lower extremities simultaneously, with contraction of the pupil and loss of consciousness. The pulse remained about 70 per minute ; the respirations, about 20 per minute, presented a peculiar jerky character, while the temperature remained about normal for six hours. The state of the pupils pointed to a lesion either in the pons or in the lateral ventricles ; but the fact that the paralysis affected both sides simultaneously excluded the latter site, and the lowness of the temperature negatived both this localisation and hæmorrhage into the pons. The sudden inco-ordination followed by an involvement of the hypo-glossal nucleus, with bi-lateral paralysis, pointed to a lesion which was not in the pons, but which involved both sides of the medulla and the motor tracts symmetrically, and this could only be satisfactorily accounted for by a hæmorrhage into the cerebellum pressing upon the fourth ventricle.

The patient lived for sixteen hours, and the temperature did not begin to rise until seven hours after the onset of the attack. It reached 104°-8° before he died.

Dr Bruce emphasised the desirability of systematic thermometry in all cases of intra-cranial hæmorrhage.

(*b*) Specimens from two cases of carbolic acid poisoning. In one where the acid was swallowed in the concentrated form, the mucous membrane of the œsophagus was coagulated and detached as a tube from the submucous membrane.

The stomach was of a deep red colour, but the rest of the alimentary tract was normal.

In the other case a large quantity of a five per cent. solution had been swallowed. The mucous membrane of the œsophagus was of a milky colour, but not detached. The stomach, duodenum and jejunum, however, showed diffuse redness and swelling of the mucous membrane, specially marked in the rugæ of the stomach, and in the valvulæ conniventes of the small intestine. The superficial layers of the mucous membrane were coagulated and desquamated.

In the first case death took place in thirteen hours, and the urine was dark coloured. The second was fatal in four hours, and the urine drawn off was normal.

(*c*) A microscopic section of the medulla oblongata from a case of syringomyelia. In this a narrow fissure passed from the neighbourhood of the extreme upper end of the central canal obliquely outwards and forwards as far as the inferior (ascending) root of the fifth nerve. It had involved the descending root of the glossopharyngeal nerve, and had atrophied many of the internal arcuate fibres from the nuclei of the posterior columns for the fillet, which was partially atrophied on the side opposite the lesion. The specimen was interesting as explaining the disturbances of sensibility in the distribution of the fifth nerve, which sometimes occurred in this disease.

2. *Drs Affleck and Bruce* exhibited (*a*) specimens of the cord from a case of locomotor ataxia in which there was marked inco-ordination of the upper as well as the lower extremities of cervical tubes. There was marked degeneration in the postero-external in addition to the usual brim in the postero-internal column.

(*b*) Scapulæ and humeri from the same case showing a

well marked Charcot's joint affection, with distension of the capsule of the joint, great thickening of the zuonal membrane, and absorption of the head of the humerus.

(*c*) Sections of the spinal cord and muscles from a case of pseudo-hypertrophic paralysis. The muscles showed the usual changes in it characteristic of the disease. The cells in the anterior cornua of the spinal cord were quite normal.

3. *Dr J. O. Affleck* exhibited a specimen of a greatly misshapen liver, the result of waxy disease. The patient from whose body the specimen was taken was a young man of 26, who was admitted into the Royal Infirmary suffering from great anæmia and weakness. The liver was found to be greatly enlarged and extended on the right side down as far as the level of the umbilicus. In the left hypochondrium there was felt a large tumour which seemed to be separate from the liver, and was strongly suggestive of the spleen. The lower limit of this tumour extended to fully an inch below the umbilicus. An examination of the blood showed about 2,000,000 red corpuscles per cmm., but only a slight increase of leucocytes. There was a fluctuating temperature, and frequent epistaxis. The whole clinical aspects of the case, as well as its history, appeared to me to resemble very much acute splenic anæmia, in which opinion I had the concurrence of Dr Robert Muir, who made repeated examinations of the patient's blood. The diagnosis seemed to lie between this and waxy disease. Arsenic gave temporary relief to most of the symptoms, but latterly the patient feeling himself getting weaker, wished to get home, where, after a few weeks, he died. On *post-mortem* examination (made by Dr Muir) it was found that the tumour in the left hypochondrium, which so strongly resembled the spleen, was the left lobe of the liver which had become enormously hypertrophied from waxy disease, and had assumed a tongue-like shape. Between it and the right lobe, which also was much enlarged, there was a large fissure, so that the two lobes seemed like separate organs. The weight of the liver was 9 lbs. The spleen was large and waxy, but it was completely concealed by the left lobe of the liver. There was also waxy disease in the kidneys and intestines. There was no tubercle nor any other ascertainable cause for the widespread amyloid change, and there was no history of syphilis.

4. *Mr Alexis Thomson* exhibited—

(*a*) HYDRONEPHROTIC KIDNEY REMOVED BY LUMBAR INCISION from woman, aged 30, who had suffered during eight years from attacks of renal pain on the right side, attended with vomiting and with irritability of the bladder. The right kidney was enlarged and cystic. With the cystoscope the bladder wall was inflamed, and pus was observed to escape from the right ureter. The kidney was removed because of the superadded pyo-nephrosis. The specimen illustrated advanced hydronephrosis, and pus was present in several of the distended calyces. The patient made a rapid and good recovery.

(*b*) A small OXALATE OF LIME CALCULUS REMOVED FROM THE RIGHT URETER by the extra peritoneal method. The stone was impacted at a point 2 inches below the origin of the ureter, and was only dislodged with difficulty after the wall of the ureter had been laid open in its long axis. An attempt was made to suture the wound in the ureter. The patient, a male, aged 40, had had five severe attacks of renal colic, with blood in the urine during a period of six weeks. Urine escaped from the wound in the loin for eight days, then ceased. The patient made a good recovery.

5. *Professor Chiene* exhibited (*a*) large URINARY CALCULUS removed from pelvis of the kidney. It filled the pelvis, and the main difficulty in its removal was the mobility of the kidney. In dilating the kidney tissue to reach the pelvis, it is best to incise the capsule at the peripheral edge of the kidney in the long axis of the organ.

(*b*) BILIARY CALCULI removed from the gall-bladder. The difficulty in the operation consisted in the small size of the gall-bladder; the impossibility before opening of bringing it forward to the anterior abdominal wall, and the necessity of careful packing before making an opening into the viscus.

(*c*) A BILIARY CALCULUS which caused complete obstruction. The patient, a female, aged 67, was seized with severe abdominal pain. On the third day the tympanitis was marked and the obstruction complete. The diagnosis was a kink or volvulus in the wall intestine. An opening was made in the middle line, and the dilated small intestine at once protruded. On handling it, a small tear took place, and a foot beyond

the tear a hard substance, evidently a calculus, was felt in the lumen of the gut; beyond the hard mass the intestine was collapsed. The calculus was easily pushed back, and removed at the accidental tear which was closed by a double row of sutures.

Where did the calculus come from? Was it lying in a Meckel's diverticulum? The patient, when a young girl, had passed gall-stones. A drawing of the calculus is given because it is difficult to see why a calculus of this size should not have passed along the intestine; possibly its irregularities may have caused irritation and spasm.

FIG. I.—A. NORMAL GUT. B. DISTENDED GUT. C. CALCULUS (ONE-THIRD ACTUAL SIZE).

(*d*) A case of abdominal contents in a fatal case of obstruction, due to adhesions of the small intestine. It was shown to illustrate the clinical fact that in appendicitis the pain may be felt on the left side, because the apex of the appendix was fixed to the parietal peritoneum over the *left* common iliac vein. There had evidently been old adhesions, and a recent abscess had caused the complete obstruction.

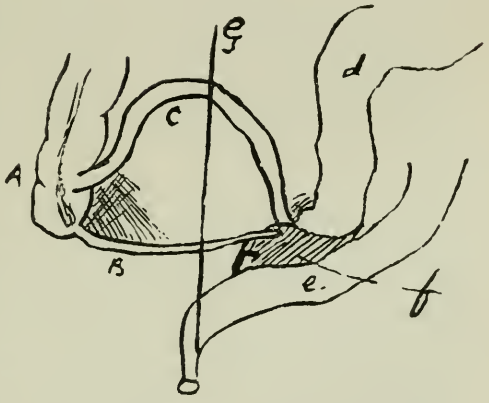


FIG II.—A. CÆCUM. B. APPENDIX. C. NORMAL SMALL INTESTINE. D. DILATED SMALL INTESTINE. E. RECTUM. F. ADHESIONS AND ABSCESS. G. MIDDLE LINE.

6. *Mr J. M. Cotterill* exhibited—

(*a*) CYLINDROMA OF BASE OF SKULL.

The patient, a male, aged 48, had been sent to Mr Cotterill by Mr George Berry.

It was found that the patient suffered from proptosis of the left eye, accompanied by paralysis of the oculomotor muscles.

There was a dense swelling in connection with the lower

eyelid and towards the malar bone. After some weeks of observation, suppuration took place in the lower eyelid, and the patient was admitted to hospital. He succumbed in a few days to septicæmia from the abscess. The tumour was found to be a cylindroma, which had originated in the fibrous tissues at the base of the skull. The tumour involved the bone as far back as the foramen magnum, and reached forward to the frontal bone, which it had perforated near the tip of the frontal lobe of the brain. The brain itself was not involved.

(*b*) MALIGNANT PUSTULE.

The patient, a man, aged 28, had been working some ten days before admission with dry hides. He presented the typical appearance of a malignant pustule, viz., the dark central slough, the surrounding crop of vesicles, the redness and tough œdema of the surrounding parts. There was also the characteristic entire absence of pain in the part.

The pustule was at once excised, together with some lymphatic glands under the chin which were affected; and in cutting through the œdematous skin, the characteristic ecchymotic patches in the cellular tissue were most observable. The patient made a good recovery.

7. *Mr David Wallace* exhibited (*a*) URIC ACID CALCULUS weighing 40 grains removed from the bladder. The calculus was not detected by sounding, but was seen on cystoscopic examination lying behind a pedunculated middle lobe of the prostate. An attempt at crushing failed as the stone could not be grasped. Suprapubic cystotomy was performed later by Professor Chiene, when it was found that the middle lobe was so mobile that it fell over the stone so as to cover it.

(*b*) A portion of a red rubber catheter ten inches in length which he removed from the bladder of a patient after its presence was detected by cystoscopy. The patient, who was paraplegic, suffered from retention, and the catheter left in the urethra overnight was in some way broken across, and passed into the bladder. The portion shown was easily grasped and removed by a litholyite of the smallest calibre.

8. *Dr C. W. Cathcart* exhibited (*a*) a solid simple EPITHELIAL TUMOUR removed from a young woman, aged 21. Eight years before its removal it had been first noticed as a

small painless pimple on the outer side of the right thigh. After growing slowly for three years it had burst, and had continued to render ever since. The discharge was generally clear, but stained the clothes yellow, sometimes the fluid was purulent, and occasionally it was blood-stained. Latterly, about half a wineglassful of the discharge had come away daily. Since it had burst the lump had continued to grow slowly larger, but occasioned no inconvenience except from the discharge. It formed a rounded swelling about the size of a chestnut at the outer side of the lower third of the thigh. The tumour was not adherent to the fascia lata. The skin over it was tightly stretched, thin, and somewhat discoloured, and in the centre showed a sharply punched-out circular ulcer about one-eighth inch in diameter, which extended deeply into the swelling, and was very liable to bleed when probed. The swelling was removed along with the skin over it. The freshly cut surface had a somewhat granular appearance—yellowish white in colour, and mottled with what seemed to be the mouths of numerous blood vessels. It was well defined all round, and its substance was breaking down in the centre where it formed the base of the ulcer.

Under the microscope, the tumour is seen to consist of epithelium, subdivided into masses of irregular size by a substance which is homogeneous in some places, and at others shows fibrillar arrangement and a few nuclei. Many spaces exist throughout the tumour substance, and some with distinct wall seem to be blood vessels. The epithelial cells are embryonic in some places like those forming the buds of a developing hair follicle ; at others they are large, and show the same kind of degeneration of the protoplasm as may be in a sebaceous gland. In fact, types of the epithelial cells can be readily recognised in a section of fœtal scalp, which was also shown in order that the two might be compared. The tumour might be considered a sebaceous adenoma.

(*b*) Two VERMIFORM APPENDICES illustrating different conditions found in recurrent appendicitis—one appendix progressing towards rupture, the other towards spontaneous cure.

The first had been removed from a girl of 18 after four attacks. Her first attack, lasting three weeks, had been in July 1896 ; the second, lasting two weeks, in October 1896 ; the third, lasting five weeks, was in July of this year ; and the

fourth, lasting three weeks, in September 1897. Since the last attack she had never been quite free from pain. Thickening was easily felt in the region of the appendix, and the part was tender to the touch. The appendix was bound down to the cæcum by numerous adhesions, which were partially organised. After removal the terminal portion of the appendix was found blocked off from the rest of the tube, and it contained a small concretion and some very fœtid pus. The presence of this isolated collection of septic matter was evidently a constant source of irritation, and perhaps at the next attack it would have burst.

The second V. appendix had been removed from a young man of 25, who described five attacks, none, however, laying him up for more than a week, and some not laying him up at all. The first was merely a sharp attack of pain lasting for a few hours, in April 1896; the second, a year afterwards, laid him up for about a week; the third, two months later, was similar to the second; the fourth consisted in more or less constant discomfort and pain while he was bicycling for a week, but he did not lay up with it; the fifth, lasting for three or four days, came on early in October. A thickening could be felt in the region of the appendix, which was tender to the touch. The appendix was found quite free from adhesions, in the abdominal cavity it was thickened and congested. On examination it is seen to be undergoing a fibroid change. The lumen is much contracted near the cæcum, and in the terminal half is obliterated.

The mucous surface in the inner half is ulcerated, and contained a plug of decomposing material, which probably kept up the chronic inflammation. After three or four more attacks, involving much loss of time and inconvenience, this appendix might have become entirely obliterated, but it was much better away. Both patients were making a satisfactory recovery.

(*c*) A LEG INJECTED AFTER AMPUTATION FOR SENILE GANGRENE OF THE GREAT TOE.

The patient, a married woman, aged 46, much emaciated. She had complained about a year before admission to the infirmary of cramping pains in her left leg. Four or five months afterwards a sore appeared on the side of the great toe. She had then intense pain in the part, and the skin of the dorsum of the toe became black. In this condition she

was admitted. The parts were thoroughly cleansed with carbolic and dressed with iodoform and the limb as far as the knee wrapped in cotton wool. After six or eight weeks no material change was observed in the foot. The pain was much less, however, but she remained very thin. The femoral artery could not be felt to pulsate below the upper part of Scarpa's triangle. At the amputation the popliteal artery after ligature did not pulsate, and the stump as a whole was very dry. The flaps were made very loose. In spite of this the mere weight of the anterior flap against the bone tended to cause ulceration, and extension of the flap was necessary during the healing process.

The injection and subsequent dissection shows that the posterior tibial artery was blocked just below the bifurcation, and that the anterior tibial artery also was blocked in its lower third. Injection had reached the posterior tibial artery below the obstruction through anastomosing branches in the leg. The presence of numerous enlarged branches coming off above the points of obstruction lead one to believe that neither of the obstructions would alone have sufficed to cause gangrene, only their combination and probably also a want of nutritive activity in the walls of all the vessels.

The obstructed part of the posterior artery contained a firm plug. Under the microscope the walls of the vessel there show considerable thickening due to fibrous tissue formation, without any fatty degeneration or calcareous infiltration. The disease, therefore, is endo-arteritis obliterans rather than atheroma. The specimen specially illustrates (1) that in cases of dry gangrene the position of the obstruction is a long way above the gangrene, and (2) that the tissues for some distance above the gangrene are very imperfectly supplied with blood, and could not easily resist sepsis or much mechanical injury of any kind, and (3) that in time the anastomosing circulation may enlarge in suitable cases and restore the vascular supply to a considerable extent.

9. *Dr T. Shennan* exhibited—

(*a*) TRI-RADIATE TAENIA SAGINATA.

The tape worm was passed by a child. The head was not obtained. What makes it a unique specimen is that, on cross-section, it is tri-radiate, two of the radiations being larger than

the third, on the free extremity of which is the genital pore, regular in successive segments.

On microscopic examination, the uterus full of ova is found to extend into all three radiations, and on surface view its branches correspond to those of taenia saginata.

(*b*) LEGS FROM DOUBLE AMPUTATION FOR SENILE GANGRENE.

The patient from whom these legs were successfully removed has been shown before the Society by Dr Miles.

They were injected through the posterior tibial with gum carmine, and after hardening, longitudinal sections were made. The result shows well the vascular line of demarcation which has extended right through the soft parts, and is beginning to attack the bone, and this at the same level. The tendons and skin below are dry and horny, and with the other soft parts on the distal side of the line of demarcation are becoming liquefied.

10. *Mr F. M. Caird* exhibited a preparation in Jores' fluid of a SCARCOMA OF THE THIGH, for which he had amputated below the trochanter minor. The patient, a female, about 30 years of age, had a growth removed from above the patella about seven years ago. Recurrence took place, and latterly it had increased with great rapidity. The tumour measured 48 inches in circumference, consisted of spindle cells with large areas of necrotic tissue and degeneration cysts. The patient made an excellent recovery.

11. *Dr C. W. Macgillivray* exhibited a specimen of an OVARIAN CYST which he removed the other day from a patient in his operating theatre, in the Royal Infirmary. It was an ordinary multilocular cyst, with adhesions pretty general over the whole anterior wall of the abdomen, these being rather firm at several places. Her temperature was normal the day after the operation, and has continued so ever since. In fact she has experienced the usual uninteresting course common to such cases. But as there still seems to linger in the minds of some people the idea that such cases have something mysterious about them and require to be placed in exceptional conditions for their recovery, it seems well to point out, that the common and ordinary hands of the general surgeon can obtain as good results as the sacred fingers of the gynecological specialist.

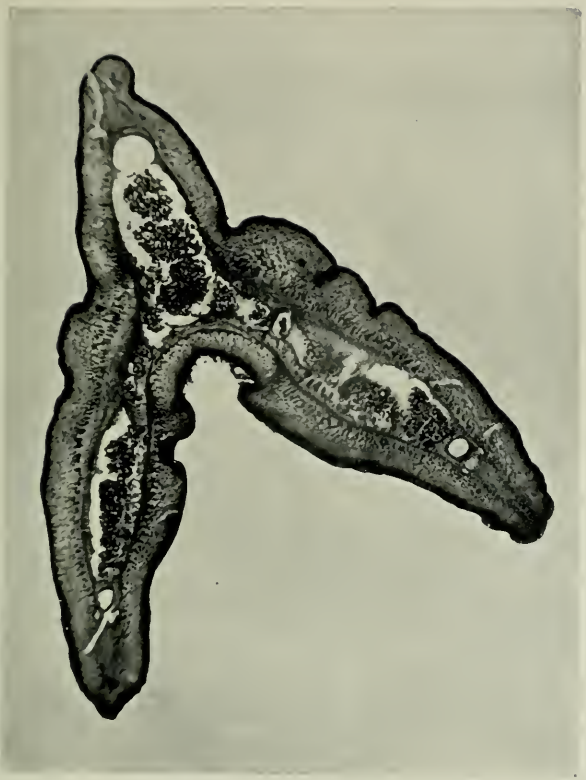


PLATE I.—TO ILLUSTRATE DR SHENNAN'S CASE OF TRI-RADIATE TÆNIA SAGINATA. (TRANSVERSE SECTION.)

Meeting IV.—January 19, 1898

SIR JOHN BATTY TUKE, *President, in the Chair*

The Society congratulated the President and Sir John Struthers on their receiving the honour of Knighthood.

I. ELECTION OF MEMBERS

The following gentlemen were elected Ordinary Members of the Society :—G. D. Darlington, M.B., C.M., and Alexander C. Ainslie, M.B., C.M.

II. EXHIBITION OF SPECIMENS

1. *Professor Annandale* exhibited—

(*a*) A large URINARY CALCULUS removed by direct incision from a new bladder which he had made some years before. The patient, a boy, æt. 7, was successfully operated upon when three years old for extroversion of the bladder, the exposed parts being completely covered over by flaps taken from the abdominal wall and scrotum. A small opening was left at the lower part for the introduction of a tube, through which the urine passed into an indiarubber bag. After the removal of the calculus, the wound was stitched and healed up thoroughly, so that the new bladder remained intact.

(*b*) The fragments of an OXALATE OF LIME CALCULUS removed from a patient 65 years of age by median lithotomy. The case was one in which apparently spontaneous fracture of the calculus had taken place in the bladder.

(*c*) A KIDNEY WITH RENAL CALCULUS IN POSITION removed from a patient. A calculus in the kidney was diagnosed, and nephro-lithotomy in the lumbar region was performed. When the stone was being removed, violent hæmorrhage took place, and as it could not be controlled by ligature or forceps, it was necessary to excise the organ. The patient made a good recovery.

(*d*) A specimen of CANCER OF THE RECTUM, removed by Kraske's method from a patient 16 years of age. About 5 inches of the rectum, including the sphincter, were removed, and the patient was almost convalescent. Mr Annandale had

met with cancer of the rectum in a patient 18 years of age, but never so young as 16.

(*e*) A specimen of OSSEOUS ANCHYLOSIS OF THE ELBOW JOINT, the result of a severe injury. As the joint was fixed in a useless position, the joint was excised. Although every care was taken, it was found on examining the parts removed that a portion of the ulnar nerve lay in a bony tunnel, and nearly 2 inches had consequently been excised with the bone. At the first dressing, two days after the operation, the wound was re-opened, the ends of the nerve found and stitched together. The result was satisfactory, for at the end of a month sensation had returned in the parts supplied by the nerve, and the movements of the joint, although not complete, were fairly good.

(*f*) Two specimens showing what Nature sometimes does in uniting fractured bones. One was an example of comminuted compound FRACTURE OF THE LOWER END OF THE FEMUR involving the knee-joint; and, notwithstanding the age of the patient—nearly 70—and a smart attack of septicæmia, relieved by anti-toxine injection, recovery took place, and amputation was performed owing to the useless condition of the limb. The patient had been a most alcoholic subject, with a great command of bad language; but after recovering well from the amputation, he became quite reformed in his habits and mild in his language.

(*g*) The second specimen illustrated a severe and comminuted FRACTURE OF THE ANKLE JOINT, with lateral displacement of the lower end of the tibia. The bones had united, but with so much deformity that the foot was useless, and amputation, through lower third of leg, was performed. The result was a good recovery.

2. *Dr John Thomson* exhibited (for Dr Playfair) the brain of the child with HEMIPARESIS AND TREMOR OF THE LEFT ARM whom he had presented to the Society at their last meeting. The child had been in the Sick Children's Hospital under Dr Playfair for some time, and had been operated on on January 12th by Dr Joseph Bell. Dr Bell trephined over the right motor centres. The intracranial tension seemed to be greatly increased, but no tumour or other local abnormality could be seen. The child died the following day.

On January 14th the *post-mortem* examination was made by Dr Welsh. There was no lesion on the surface of the

brain except a few small grey tubercles along the sylvian fissure, and marked thickening of the pia-arachnoid over the base.

On cutting into the brain, a large rounded, sharply demarcated, caseus mass was found in the substance of the right hemisphere. This tumour was 1½ inches in diameter in some directions, and about 2 inches in others. There was a narrow layer of softened gelatinous brain substance behind it. The tumour occupied the site of the head of the caudate nucleus, the greater part of the lenticular nucleus and the anterior limb, and genu of the internal capsule. All of these parts were destroyed by it. The optic thalamus seemed to have almost entirely escaped.

The upper extremity of the mass only reached to the roof of the lateral ventricle. Its outer side lay immediately internal to the grey matter of the Island of Reil, and was 1·5 cm. from the surface of the brain. At all other points the tumour was more deeply placed. The inner surface of the tumour reached to within 2 mm. of the mesial plane. Its posterior surface was just in front of a vertical line through the middle of the fissure of Rolando.

There was no other caseous nodule in the brain or spinal cord.

3. *Dr Alexander Bruce* (on behalf of Sir Thomas Grainger Stewart) exhibited a specimen of ANEURISM that had ruptured into the left bronchus. The patient, who had been exposed to great privation previous to his admission to hospital, appeared at first to have a left-sided pneumonia with pleural effusion. Aspiration, repeated on four occasions, failed to remove any fluid from the pleural sac. The breath sounds, previously faint, gradually disappeared altogether from the lower lobe of the left lung, while the resonance became almost lost. At the apex in front and behind the percussion note, at first skodaic, became ultimately absolutely dull, and the breath sounds became gradually more and more faint. There was no cardiac displacement, and no dulness on percussion to the right of the sternum, nor were there any cardiac or arterial murmurs. During his stay in hospital there was an irregular pyrexia, but his condition had apparently begun to improve when a copious hæmorrhage from the lungs was the cause of his sudden death.

Post-mortem.—The left lung was found generally adherent to, and so firmly, that it had to be dissected away from the chest wall and diaphragm. The lung showed a chronic interstitial pneumonic with numerous small cavities, especially in the lower lobe. The first and second portions of the aorta were dilated, but mainly towards the left. A secondary sac, about the size of a walnut, projected from the ascending arch of the aorta, and had evidently first compressed, and subsequently ruptured, into the main branches of the left lung.

4. *Dr T. Shennan* exhibited—

(*a*) LARYNX, TRACHEA, AND LUNGS FROM A CASE OF DIPHTHERIA.

The patient was a woman of 40 years, who became ill on a Friday. Diagnosis of laryngitis was made. She was brought into Leith Hospital at one o'clock P.M. on the following Sunday, and died rather suddenly at three o'clock.

The preparation showed the diphtheritic membrane loosely adherent to the back of the epiglottis, firmly adherent in the larynx, easily separated below that. In the bronchi the membrane still formed practically a complete tube; and the lung on one side was dissected to show it lining the branching bronchi, even in those of about $\frac{1}{12}$ in. in diameter, the usual condition being that the membrane occurs in patches in the bronchi.

On culture a pure and abundant growth of the long form of the Klebs-Lœffler bacillus was found, which is of interest in connection with the great virulence of the disease in this case.

The organs were preserved by Jores' Formalin method, so that the natural colours were in great part preserved.

(*b*) CASE OF IMPACTED GALL STONES IN THE COMMON BILE DUCT.

The hepatic artery was injected with starch, and the bile duct dissected out and shown in its relations to neighbouring structures.

The common bile duct was dilated to 4 inches diameter, walls thickened, and considerable infiltration surrounding. The bile ducts and cystic ducts were also thickened and dilated.

The gall bladder was dilated.

The common bile duct below the entrance of the cystic duct contained five stones, the lower one lying against the constricted opening into the duodenum.

There had been only slight jaundice, and operation was deemed inadvisable on account of the great weakness and frailty of the patient.

5. *Dr Harry Rainy* exhibited a specimen of ACHONDROPLASIA. The fœtus was a male one, and was born at the eighth month. The mother had previously given birth to healthy children, and there was no evidence of syphilitic heredity. The infant breathed feebly for about an hour after birth, but it was impossible to keep it alive longer, though every effort was made to do so. The case is figured in Dr John Thomson's paper "On the Diagnosis and Prognosis of Certain Forms of Imbecility in Infancy," read at this meeting. See page 89.

III. Original Communications

1. DRAINAGE THROUGH THE FOURTH VENTRICLE IN A CASE OF ACQUIRED HYDROCEPHALUS DUE TO CHRONIC NON-TUBERCULAR BASAL MENINGITIS

By Alexander Bruce, M.D., F.R.C.P.Ed., Assistant Physician, Royal Infirmary, Edinburgh; and Harold J. Stiles, F.R.C.S.Ed., Surgeon, Royal Hospital for Sick Children, Edinburgh

Ever since first reading the classical account by Hilton in his "Rest and Pain" on acquired hydrocephalus consequent upon closure of the foramen of Magendie, it has appeared to me that this condition was capable of being relieved by giving free exit to the accumulated fluid within the ventricles, and further, that this could best be done by an operation which gave the fullest access to the roof of the fourth ventricle. By this means the surgeon would be enabled to determine the exact cause of the obstruction, and by removing or overcoming it to re-establish, as nearly as possible, the natural exit for the cerebro-spinal fluid, without any injury to brain substance, as is necessarily involved when the lateral ventricles are aspirated.

The examination of a number of cases *post-mortem* has shown me that the most frequent causes of the obstruction are (*Med. Chir. Trans.*, 1897, p. 245):—

1. Simple fibrous closure of the foramen of Magendie.
2. The adhesion of the surfaces of the tonsils of the

cerebellum to each other and to the margins of the fourth ventricle.

3. Cysts formed by adhesion between the arachnoid and pia at the postero-inferior aspect of the cerebellum, in which case the foramen of Magendie may be open.

I have not, however, until recently had an opportunity of testing the value of directly operating on the roof of the fourth ventricle in a suitable case. In the meantime other surgeons, Parkin, Waterhouse, and (in Dr Barlow's case) Ballance have attacked the problem successfully in other ways, which will be referred to by Mr Stiles in his account of the technique of the operation. As the grounds for the diagnosis of the disease have been already fully discussed at a recent meeting of the Society, and as the object of this paper is mainly to draw attention to the ease with which the fourth ventricle can be reached and lesions in its roof dealt with, I shall content myself with a brief *resumé* of the symptoms presented by the patient under my care.

The patient was a delicate, greatly emaciated girl, aged thirteen, presenting well-marked signs of congenital syphilis in the form of pegged teeth, flattened bridge of nose, and traces of interstitial keratitis in both eyes. She had always been weakly, and a year previous to her admission to hospital, an operation had been performed on her left femur by Mr Duncan, the nature of which has not been ascertained. She was admitted to hospital (Ward XXV.) on the 10th of August, complaining of headache and backache. No definite account of her symptoms on the immediately preceding days could be obtained. After four days, as her constant cries disturbed the other patients, she had to be transferred to Ward VI. There was no history of tuberculosis in the family. When examined, she was found to have marked head retraction, widely dilated and equal pupils which, however, reacted to light. Her temperature was 101·8°, her pulse 100, and the respirations 26. Her respiratory, circulatory, and hæmopoietic systems were practically normal. Her tongue was dry, covered with a thick brownish fur. It could be protruded straight and was freely movable in all directions. She clamoured continually for food. No sooner was one meal finished than she asked for the next, with an almost unceasing importunity. The bladder was distended and the urine dribbled away constantly. It was found

to contain albumen and pus. There was a bedsore on her back which was evidently painful. She lay with her head rigidly retracted, the legs and arms also more or less rigid, the former being kept flexed, so that it was difficult to elicit a knee-jerk. The plantar reflexes were, however, present on both sides. The limbs could be moved, and seemed equally weak on both sides. The other organic reflexes were normal. There was a corneal ulcer on the right eye. Although the pupil reacted to light, it was difficult to test her power of vision, owing to the impossibility of fixing her attention; but further examinations at intervals made it perfectly certain that her power of vision gradually diminished. There was no optic neuritis. Her hearing, on the other hand, was abnormally acute, and her speech was in no way impaired. Replies were readily given to any question addressed to her. The further progress of the case showed an irregularly oscillating pyrexia seldom exceeding 103°; continued retraction of the head, which varied somewhat from day to day; retention of her mental power with increasing blindness and weakness of the limbs; some difficulty in swallowing, apparently due to the head retraction. About the 23rd of August she seemed unable to move her left arm and leg. The muscles of both eye-balls were paralysed, with the exception of the right external rectus, which maintained a constant spasmodic movement, causing nystagmus and external strabismus. A considerable degree of cyanosis developed.

The condition remained much the same till the 7th of September, when she became distinctly weaker. The cyanosis increased. At the Ward visit she was found to have become unconscious, and to be having a rigor. It was ascertained from the nurse that a similar rigor had occurred on the previous afternoon.

As the symptoms pointed, with tolerable definiteness, to a chronic, syphilitic, basal meningitis with secondary hydrocephalus, and, as the girl's condition seemed to have become critical, it was decided, after consultation with Mr Stiles, that he should endeavour to relieve the distention of the ventricles by trephining the occipital bone, so as to reach the fourth ventricle and allow the escape of the excess of cerebro-spinal fluid.

Surgical Treatment and Remarks, by Mr Stiles

On the 7th of September I was asked by my colleague, Dr Bruce, to see the patient with a view to operative interfer-

ence. Dr Bruce pointed out that the patient's symptoms were probably due to closure of the communication between the ventricles and the infra-cerebellar subarachnoid space. He desired, if it were feasible, that I should explore the fourth ventricle, with the object of re-establishing this communication. I was aware that this had been done successfully by Parkin and by Waterhouse; and from a preparation of a child's head which I possessed, showing a dissection of the fourth ventricle after removing a wedge from the occipital bone, I was satisfied that the operation ought not to present any special difficulty.

As the patient's condition had become critical within the last twenty-four hours, it was decided to operate at once. Chloroform having been administered, the patient was placed in the prone position, and (all the retraction having disappeared) the head was flexed upon the sternum in order to give access to the field of operation. A horse-shoe shaped incision, passing at once down to the bone, was carried from a little behind the apex of one mastoid process to a corresponding point on the opposite side, the highest part of the curve reaching a little below the external occipital protuberance. The integuments, together with the muscles at the nape of the neck, were rapidly dissected down from the occipital bone until the finger felt the posterior border of the foramen magnum; pressure forceps were applied to the occipital arteries, and to several bleeding points in the nuchal muscles. The flap having been held down out of the way by means of a stitch passing through its apex and the skin of the neck below, a $\frac{3}{4}$-inch trephine was applied to the occipital bone in the mesial plane, a little above the foramen magnum. The disk of bone removed was found to include a small segment of the foramen magnum. The dura bulged, and did not pulsate. The trephine opening was enlarged in the transverse direction by gouge forceps. A curved needle, threaded with fine silk, was then passed through the dura at the upper and lower parts of the exposed membrane, so as to include and ligature the small sinus in the falx cerebelli. The dura was incised transversely between the ligatures, and additional vertical incisions having been made, the two dural flaps were turned back so as to expose the arachnoid covering the posterior extremities of the cerebellar tonsils and its reflection from them downwards over the lower part of the medulla. The arachnoid, thus exposed, was found to be slightly thickened

and more opaque than normal. The opposed surfaces of the tonsils, which were bound together by subarachnoid adhesions, were then separated by insinuating between them two curved periosteum separators. Immediately this was done several ounces of clear cerebro-spinal fluid spurted out, and continued to well up copiously whenever the child retched or strained in any way. After the fluid had escaped, the posterior medullary velum, the choroid flexuses, the foramen of Magendie, and the lower part of the medulla could all be distinctly seen. The dura was not stitched, neither was any bone replaced. The flap was sutured into position by means of interrupted stitches of silkworm gut. The fourth ventricle having been so freely opened up, it was not deemed necessary to provide specially for drainage by means of a tube or horsehair. The sudden escape of so large a quantity of cerebro-spinal fluid produced no excessive shock or other alarming symptom, nor did I expect that it would from what I have seen of the results of withdrawing many ounces of this fluid in tapping cases of hydrocephalus and spina bifida.

The patient bore the operation well, and soon after being put back to bed was able to answer questions, expressed a desire for food, and talked about what she would like for dinner. There was less complaint of headache. The temperature fell to normal immediately after the operation, and remained so for four days. On the fourth day it ran up to 101°, and subsequently it rose and fell at irregular intervals. Shortly before death, which took place on the nineteenth day, the temperature reached 106·2°. The pulse and respirations were not materially influenced by the operation. The mental condition maintained the improvement to the end; the patient, however, became more and more emaciated, and ulceration of the cornea, which had occurred before the operation, set in again in an aggravated form a few days before death.

A striking feature of the case was the very large quantity of cerebro-spinal fluid which drained from the wound: large dressings were soaked through in from twelve to twenty-four hours.

On the tenth day, the wound being healed throughout, the stitches were removed in the hope that by so doing the drainage, which escaped by the stitch punctures, would become less abundant. The fluid, however, accumulated under the flap, and

when next the case was dressed it was found that a small sinus had formed opposite the centre of the wound, and through it the cerebro-spinal fluid continued to drain away. That cerebro-spinal fluid is, under certain conditions, very rapidly produced, is well known. As an illustration of this I may refer to the case of an infant with a large spina bifida which I lately had under treatment. On each occasion after tapping and drawing off as much as ten ounces it was found that the sac filled again in less than twenty-four hours. The child gradually became hydrocephalic, and it was evident, therefore, that the cerebral ventricles took a large share in the production of the fluid which filled the sac of the spina bifida.

The question arises—Was the drainage in the case of our patient too free, and would the patient have recovered if we had been able to control it? It seems clear that if the morbid condition be such as to produce a large excess of fluid, an efficient drain must be established, otherwise the ventricles will again dilate, and the symptoms which called for the operation will recur.

At the *post-mortem* examination, the ventricles of the brain were found greatly dilated, and the convolutions flattened. There was a well-marked basal meningitis, extending from the third nerve backwards under the pons and medulla. The foramen of Magendie was found to be patent. The accumulation of cerebro-spinal fluid was due to an adhesion of the two tonsils of the cerebellum to each other and to the sides of the medulla. The separation of the two tonsils, which was brought about by the operation, had remained permanent. The other organs showed a complete absence of tubercle. There was a small pulmonary apoplexy in the lower part of the lower lobe of the left lung. The right kidney showed a recent breaking-down infarct (? abscess) about the middle of the organ.

As comparatively few cases have been reported in which the fourth ventricle has been attacked, I may briefly refer to them.

The first case appears to be that of Mr Alfred Parkin, of Hull, who, on the 9th of April 1893, opened the basal sub-arachnoid cavity for the purpose of relieving intracranial pressure in a child aged four and a half years, who was in the comatose stage of tubercular meningitis. The procedure he adopted was as follows:—A circle of bone was removed with a ¾-inch trephine from the right cerebellar fossa of the occipital

bone, and the opening was enlarged in a downward direction by Hoffmann's forceps. After incising the dura, a curved probe was passed along the under surface of the cerebellum towards the fourth ventricle. Two to three ounces of clear fluid gushed out. A very fine drainage tube was inserted into the subarachnoid space, and the dura and scalp both sutured. The child died sixteen hours after the operation, free drainage being maintained to the end. The author recommended the same operation for other conditions associated with intracranial pressure, the result of excess of cerebro-spinal fluid. He regards this operation as preferable to lumbar drainage.

Since then the following cases have been recorded, and no doubt others have been operated upon, although not published.

1. A child, aged eleven months, suffering from congenital hydrocephalus, operated on in July of the same year, again by Mr Parkin. In this case a horsehair drain was used, and the drainage, though never very abundant, was sufficient to prevent tension of the anterior fontanelle. The drain was removed on the eighteenth day—three days after the fluid had ceased to flow. On October the 30th of the same year, that is to say three months later, the child was reported to be in excellent health, and to have gained in intelligence and weight.

2. A child, aged five years, presenting all the symptoms of tubercular meningitis, was operated upon by Mr Waterhouse on October 26th, 1893, with resulting recovery. He trephined over the left cerebellar fossa, and passed a bent probe inwards between the cerebellum and the arachnoid. After striking the falx cerebelli, the instrument was rotated forwards into the subarachnoid space, between the cerebellum and the medulla. Some drachms of clear fluid escaped. A drainage tube was inserted, the dura sutured, and fragments of the bone replaced. As in our own case, large dressings were repeatedly soaked with cerebro-spinal fluid. Rather more than a month later the patient was reported to be quite well and able to sit up to meals in the ward. There was no longer any headache, and the temperature had been normal since November 18th.

3. An infant, aged five months, suffering from congenital hydrocephalus, operated on by Mr Parkin, September 3rd, 1895. The child died on the sixth day from hyperpyrexia, which Mr Parkin attributed to excessive escape of cerebro-spinal fluid.

4. Chronic hydrocephalus in a child aged three and a half years, operated on by Mr Parkin, March 17th, 1894. Operation as before, except that a silk drain was introduced into the cavity of the fourth ventricle, and kept *in situ* for twenty-eight days. On June 8th, 1895 (ten months later), the patient was reported to have gained in intelligence, and the bodily increase in size, with absence of cranial increase, had almost removed the disparity which previously existed.

5. A man, aged eighteen years, presenting symptoms of a cerebellar tumour, was operated upon by Mr Thomas of Liverpool, on April 17th, 1895. The right cerebellar fossa was trephined close to the middle line. No tumour was discovered, but on passing the finger towards the fourth ventricle a soft bulging immediately gave way, allowing the posterior part of the medulla to be felt. On withdrawing the finger, a quantity of clear fluid gushed out. Mr Thomas concluded that an internal hydrocephalus had been relieved. The dura was sutured; no drain was placed within the cranial cavity. The headache was immediately relieved. Two months later, the patient expressed himself as quite cured, and was able to go to a convalescent home.

Mr Waterhouse, in reply to a letter asking him for a reference to his paper, informs me that since it was published he has opened the inferior medullary velum in eight cases: in three for tubercular meningitis, all of which died; in one for chronic non-tubercular basal meningitis, with resulting cure; in four for chronic internal hydrocephalus, two of which remained *in statu quo*, one died four months later, and in the remaining one marked improvement resulted.

When I performed the operation upon Dr Bruce's patient, I was not familiar with the details of the operation as performed by Parkin and Waterhouse. Selecting what we considered to be the most direct route whereby to expose the fourth ventricle, the occipital bone was trephined in the mesial plane, close above the foramen magnum. It is true that the bone is here thicker where the internal occipital crest is encountered than it is over the cerebella fossæ, and in the former situation the falx cerebelli has to be dealt with; but these anatomical considerations are of no importance, and are minor disadvantages, which are more than counterbalanced by the advantage which is afforded by the free and visible access which the median operation gives to

the fourth ventricle. Moreover, the median operation allows the cerebellar tonsils to be separated should they be glued to one another and to the posterior substance of the medulla by inflammatory adhesions.

Of the fifteen cases here collated, five are claimed as having been cured. It is difficult, however, to say how far Mr Parkin's two cases of congenital hydrocephalus are to be regarded as cures; sufficient time had hardly elapsed between the operation and the report of the cases, to enable one to say whether or not the patients were really cured, or merely improved. It is to be noted that Mr Waterhouse does not speak of a cure in any one of the four cases of congenital hydrocephalus upon which he operated. Although he regarded his first case as one of tubercular meningitis, proof of this must be held to be wanting in the absence of positive results following experimental inoculation. If this case was not really tubercular, then in none of the cases of tubercular meningitis has recovery followed the operation; in none, however, could it be said that death resulted from it. As all those who have performed the operation agree that it is perfectly simple, it seems to us that it is deserving of further trial in tubercular meningitis, and in all those morbid conditions where increased intracranial tension is threatening the life of the patient.

If Mr Waterhouse's first case was not really tubercular, then his two recoveries were both cases of simple chronic basal meningitis. It is especially in these cases of acquired hydrocephalous, due to chronic non-tubercular basal meningitis, that we are disposed to urge our operation, and we would advise that it be undertaken as soon as the diagnosis can be made.

2. ON THE DIAGNOSIS AND PROGNOSIS OF CERTAIN FORMS OF IMBECILITY IN INFANCY.

By JOHN THOMSON, M.D., F.R.C.P.Ed., Extra Physician to the Royal Hospital for Sick Children.

THE diagnosis and prognosis of mental deficiency or imbecility in infants often forms one of the hardest as well as one of the most interesting questions of a Children's Hospital Out-patient Room. It is on this account that I venture to bring the subject before you this evening in the form of a few

remarks and a demonstration of photographs illustrating some of the commoner varieties of disease which lead to this condition in early life. Whatever grade of mental defect is present, it is of course best regarded as a symptom rather than a disease; and the questions presented to us in the first place, are, What is the disease which is causing these mental phenomena, and how is its future course likely to affect their continuance and character?

The classification of the various pathological conditions which give rise to imbecility and idiocy is a very difficult matter. I shall not therefore attempt to enter into it here but shall merely make a few remarks on some of the best known and most distinct groups in which feeble-minded children are usually placed. It is mainly those which present marked bodily malformations of which I wish to speak, and what I have to say refers mainly to young infants (*i.e.*, from birth to three years old). While most of it is drawn from observation of my own cases, I have to acknowledge also the great help and instruction I have derived from the writings of Dr Langdon Down and Dr Shuttleworth, and especially from Dr Ireland's classical work on "Idiocy and Imbecility."

Many of the mentally defective infants we meet with, present none of the grosser physical defects which we are about to consider; in these we may have to found our diagnosis entirely on the degree of delay in the development of the bodily and mental powers present, taken along with surrounding circumstances. Thus we must think of mental deficiency as possibly present if a child is very long of learning to hold up his head, to sit, to use his hands, to stand, and to walk, and especially if he does not show the natural desire of the healthy infant to exercise all his developing motor faculties. Or again, if he is backward in noticing objects and in responding to the smile and voice of his mother and nurse; and at a later stage, backward in speaking.

Backwardness in the acquisition of these natural actions and gifts may be due, of course, merely to temporary debility accompanying or following bodily illness, and it often is so. If, however, the degree to which it is present is marked, and there is no history of illness or sign of debility to account for any slowness of this sort, we will probably be right in attributing it to mental defect.

1. *Microcephalus*

The first group to which I wish to direct your attention is a small one, that, namely, of microcephalic idiots. Noticeably small heads are, of course, very often seen in imbeciles of various kinds, especially after the first year or two of life, owing to the damaged brain not having grown as it should have done, but true microcephalic cases are comparatively seldom met with.

In these cases (figs. 1 and 2), the head is strikingly small even in very early life, and it has also a peculiar shape. The forehead is very narrow and receding, the vertex pointed and the occiput flat. The circumference of the cranium is much below the normal, thus in one baby aged 4¾ months

FIG. 1.—MICROCEPHALUS. GIRL, ÆT. 4¾ MONTHS.

FIG. 2.—MICROCEPHALUS. GIRL, ÆT. 2 YEARS.

(fig. 1) it was 13¼ instead of 15½ or 16 inches, as it should have been; in another of two years (fig. 2), 15½ instead of about 18½ inches. The palate is generally high and deformed. A very characteristic symptom in early infancy is the abnormally early closure of the anterior fontanelle. I do not know how soon this may take place, but I have seen it quite closed in one child at 4¾ months and in another at 5½ months. So far as I know, closure of the fontanelle before the seventh month is always due to microcephalus. The teeth appear about the normal time.

Apart from the cranium, the child's body is well made. The features (in infancy, at least, when their lack of expression is not so noticeable as later) are usually rather pretty. The ears are large and well-formed. The hair fine and plentiful.

In infancy the child is well-nourished and of a fair size, although he is usually undergrown in later life. His limbs are large and his muscles are strong, so that his grip is powerful although unintelligent. He is more or less late of walking, and when able to do so has a peculiar gait, carrying his head in front of him in an even more marked way than other imbeciles do. Many of these children have convulsions. Mentally they are very dull indeed, and quite apathetic unless they are hungry.

The prognosis as to life is not very good ; a considerable proportion of these children die before they are old enough to be admitted into an institution. When they survive infancy they may live to a fair age, as in the case of the so-called " Aztecs," who have been under the observation of the medical profession and of the public for at least forty years. They are, however, capable of little mental improvement under education, although it makes them much less mischievous and troublesome. There seems to be no evidence that microcephalic children are ever really benefited by any surgical operation undertaken to enlarge the capacity of their cranium.

2. *Chronic Hydrocephalus*

This is not a very uncommon cause of mental defect. It may be congenital or it may arise soon after birth. Not infrequently it follows non-tubercular meningitis.

FIG. 3. — CHRONIC HYDROCEPHALUS. BOY, ÆT. 9 MONTHS.

In hydrocephalus the head is enlarged and globular. The eyeballs have generally the characteristic downward direction (fig. 3), so that the border of the lower eyelid tends to cross the pupil and the sclerotic is seen above the iris to a more than usual extent. The fontanelles and sutures are abnormally large, and they remain open very long, the anterior fontanelle being often patent up to four or five years of age. The teeth appear early, but the child is generally long of walking. He is very often subject to fits. The body is usually small and weakly.

Mentally, the defect is not very noticeable, and, in slight cases, the intellect may never be appreciably affected. Even in extreme cases the children show a surprising amount of intellect, considering the condition of their brains. In after life, children who are imbecile through hydrocephalus are often capable of a considerable amount of education, and they are usually gentle and amiable in disposition.

3. *Cerebral Infantile Paralysis*

In any form of cerebral paralysis there may be some degree of mental impairment. It is most likely to be severe in those cases of diplegia in which meningeal hæmorrhage at the time of birth has led to atrophic changes in a large area of the cortex on both sides of the brain (fig. 4). There may, however, be considerable mental defect in cases of even comparatively slight hemiplegia or paraplegia, and in those who are by no means idiotic or even imbecile there is often a certain degree of mental instability and emotional weakness or a tendency to epileptic fits.

FIG. 4.—CONGENITAL SPASTIC DIPLEGIA. GIRL, AGED 14 MONTHS.

I need not attempt to enter fully into the diagnosis of these cases of paralysis, but will only say that their most striking feature is the stiffness and spastic condition of the limbs, and this is very obvious even in early infancy. As the children grow older, if the cerebral lesions are extensive, the head fails to enlarge in proportion to the body; but there is no tendency to premature closure of the fontanelle. These patients frequently squint, and they often make a snorting noise in breathing. In those of them who are able to speak there is often great difficulty in articulation.

When the lesion is at all extensive, the general growth of

the body is apt ultimately to suffer considerably, so that, in older childhood, the patients often look much younger than they really are. The general vitality is also usually lessened, as in most other forms of idiocy, so that most of the severe cases die in childhood, but occasionally even they live, with care, to a good age.

The capacity for education varies, of course, with the extent of the lesion. While the most advanced cases are very hopeless idiots, many of the less severe ones are capable of much more education than their stiff limbs and defective speech would lead you to expect.

4. *Mongolian or Kalmuc Imbeciles*

A very interesting and quite distinct class is formed by those children who are now universally known by the somewhat unfortunate name of "Mongolian or Kalmuc imbeciles," owing to a curious resemblance, pointed out by the late Dr Langdon Down, between their physiognomy and that of members of these races (figs. 5-10). They are comparatively common (forming, according to Shuttleworth, 5 per cent. of all imbeciles), and anyone familiar with their characteristic appearance can readily diagnose them even at the time of birth. If they are fairly well nourished they are especially easy to recognise. If thin or very weakly their appearance is not so strikingly peculiar. They are very often mistaken for cretins.

The anatomical peculiarities of these children are numerous and distinctive. The features are small, short and rounded; and generally, like the rest of the body, they are reddish in tint. The head also is short (brachycephalic) and rounded, and strikingly devoid of eminences. The eyes are often rather near one another, and, in most cases, the axes of the palpebral fissures are abnormally oblique. In a large proportion also there is a marked development of the so-called epicanthic fold of skin at the inner angle of the eye. The tongue almost always protrudes a little when the child is at rest, being apparently too large for the mouth. In older children the mucous membrane of the tongue is said to be always fissured more or less deeply, and I have found this to be the case in all the children over four years that I have seen. I have never seen any fissuring, however, in a child under two years (out of eleven cases), although in one a little over two it seemed to be just

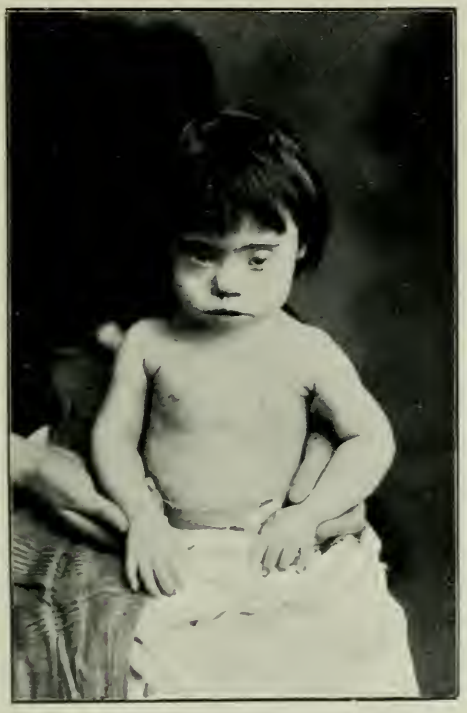


FIG. 5.—MONGOLIAN IMBECILITY. GIRL, ÆT. 16 MONTHS.

PLATE II.—TO ILLUSTRATE DR THOMSON'S PAPER.

FIG. 6.—BOY, ÆT. 18 MONTHS.

FIG. 7.—GIRL, ÆT. 2 YEARS.

FIG. 8.—GIRL, ÆT. 3 MONTHS.

FIG. 9.—BOY, ÆT. 8 MONTHS.

FIG. 10.—GIRL, ÆT. $6\frac{1}{2}$ MONTHS.

MONGOLIAN IMBECILITY.

PLATE III.—TO ILLUSTRATE DR THOMSON'S PAPER.

commencing. There is nearly always a tendency to suck the tongue. Snoring, deficient nose-breathing, and other symptoms of adenoids are often present. The hair is usually scanty and dry. In infancy the skin is generally very soft, although it becomes dry and harsh in later life. In three cases I have found congenital heart-disease. The state of the general nutrition is usually tolerably good; those who survive till puberty are apt to get very fat about that time.

The limbs are soft and small-boned and they have unusually lax ligaments at all their joints, so that they can be readily hyper-extended. The hands are most characteristic and markedly different from those of cretin babies. The wrist and the metacarpal portion of the hand are small, and the latter very soft, from the smallness of the bones and the yielding character of the ligaments. The fingers are usually thick for the size of the hand, but taper at the tip, and are not so square-pointed as those of cretins. The little finger is generally dwarfed and curved towards the ring finger. As Dr Telford-Smith has pointed out,[1] the shortness of the little finger may be due to unnatural smallness of its middle phalanx; but this is not always the case.

The development is slow in many ways. Dentition sometimes begins at the usual time, but more frequently it is delayed. The first teeth appeared in several of my cases when the child was about fifteen months old. The muscular movements are slow of being acquired, thus the baby often does not hold up his head till the sixth or even ninth month, or sit till the end of the first year. Walking is generally learned during the third year. The grasp is usually feeble. Speech is learned late and very slowly, and it is guttural and indistinct. The disposition is usually bright and lively, and the child is often quick at doing little things which are learned by imitation, although in other respects he is distinctly backward in his mental acquirements. The body-growth is almost always backward, even from birth, so that at eight months the infant looks not more than six months old, and at eighteen months about a year. The general strength also is much below the average, so that acute diseases of all kinds are badly borne—slight bronchitis, for example, being very apt to lead to fatal atelectasis or broncho-pneumonia. The

[1] *Pediatrics*, p. 315, Oct. 1896.

digestion is often very feeble. Fits occasionally occur although not so often as in most other forms of imbecility. The liability of these children to succumb to disease—especially to tuberculosis and broncho-pneumonia—is such that most of them die in infancy or early childhood. Only a small portion reach adolescence or adult life.

In giving a prognosis as to the child's future capabilities we have to remember that these children always improve up to a certain point when time is allowed for their retarded development, and sometimes make what for them may be considered fairly satisfactory progress under careful training. We can tell for certain, however, from their very earliest infancy that they will never be more than imbeciles of a somewhat low grade. But we can also predict that they will almost certainly be cleanly in their habits, amiable and affectionate in their disposition, and capable, with care, of considerable improvement.

When they become unwieldily fat at or after puberty, the administration of thyroid substance may be of considerable use. Under other circumstances, however, it is, so far as my experience goes, of no value whatever.

5. *Cretinism*

We have seen and heard so much about cretinism in recent years that it would be quite superfluous for me to waste the time of the Society by giving here any systematic description of the disease. I propose to confine myself therefore to a few remarks on its diagnosis mainly in early infancy (figs. 11 and 12).

Cases of greatly deformed new-born cretin babies have been described with large fatty tumours and other characteristic appearances. These, however, in this country, must be very rare indeed. As a rule, even, in cases which afterwards present the severest type of the disease, the child's relatives notice little amiss with him until he is several months old. Thus in a case published by Dr Byrom Bramwell,[1] which at sixteen years old showed more marked deformity than any other case I have seen recorded, there was nothing noticed wrong with the child until she was nine months old, except that her tongue was too big and her skin more mottled than

[1] *Edin. Hosp. Reports*, vol. iii. p. 203, 1895.

PLATE IV.—TO ILLUSTRATE DR THOMSON'S PAPER.

FIG. 11.—BOY, ÆT. 7½ MONTHS.

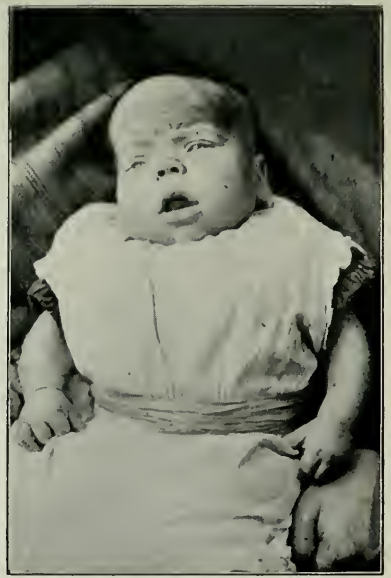


FIG. 12.—GIRL, ÆT. 15 MONTHS.

CRETINISM.

FIG. 13.—ACHONDROPLASIA. BOY, ÆT. 5 MONTHS.

PLATE V.—TO ILLUSTRATE DR THOMSON'S PAPER.

natural. In the case of the infant cretins I have had to do with, the mother's complaints have been that the child was too quiet and dull, did not cry out like other children, and scarcely ever laughed ; and in all cases there has been extreme constipation. In no case, however, was any physical defect remarked on, except sometimes the large size of the tongue. This tardy development of the characteristic signs of the disease makes it especially important that we should be on the outlook to mark its earliest indications, for there is good reason to believe that the ultimate degree of improvement under treatment varies directly with the earliness of the age at which it is begun.

I need not speak of the diagnosis between cretins and microcephalic, hydrocephalic and paralytic idiots, the differences are so great and the real resemblances so few. The two classes of deformed children which are most apt to be mistaken for cretins are the *Mongolian imbeciles* we have just been considering, and those who are suffering from *achondroplasia*, the morbid condition which is still often called "fœtal rickets," although it is really quite as distinct from that disease as from cretinism.

FIG. 14.—ACHONDROPLASIA. STILL-BORN INFANT.

Achondroplasia is a *fœtal* disease in which there is "an absence, arrest or perversion of the normal process of endochondral ossification of the most definite and universal character in every element of the skeleton in which the process normally takes place during intra-uterine life."[1] Children affected by it are therefore born with a peculiar deformity (figs. 13 and 14) which persists during life. Their heads are relatively rather large, their bodies narrow about the chest although otherwise

[1] Symington and A. Thomson, *Lab. Rep. Roy. Coll. Phys. Ed.*, vol. iv. p. 238, 1892.

I have to thank Dr Ireland for the use of fig. 5 and Dr Rainy for the photograph reproduced in fig. 14.

tolerably normal. Their extremities are very abnormally short—often only half the proper length. The great majority of these children are, for some unexplained reason, premature and stillborn, or they die within a few days of birth. In those who survive, however, there does not seem to be any mental defect, nor, indeed, any bodily one which is not secondary to the main bone-lesion. They do not show any symptoms of cretinism or imbecility in later life, but grow up to be ordinarily intelligent short-limbed dwarfs[1] such as we often may see exhibited in the side-shows of popular entertainments.

Typical achondroplasic dwarfs have at birth a very considerable resemblance to cretins. Thus they have a relatively large broad head, a marked pug nose with usually a distinct depression at its root, and often some protrusion of the tongue out of the mouth; also their limbs, including the hands and feet, are thick and extremely short. But you will observe that this resemblance is not to cretin babies so much as to those who are old enough to have developed to the full all the characteristic cretinous deformities. The limbs of cretins, for example, are not very small as compared with their bodies in early infancy; the great disproportion arises only in the course of years.

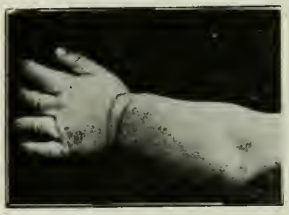


FIG. 15.—ACHONDROPLASIA. HAND OF BOY, ÆT. 5 MONTHS.

We may further notice that the skin in the achondroplasic baby is soft and natural, the hair fine and plentiful, the temperature not subnormal, and his mental condition, if he survive, like that of other infants. His thyroid also may usually be felt of a normal size, and he does not develop fatty supra-clavicular pads. His hands, although like those of cretins in being short and broad, with crumpled, apparently redundant skin, differ distinctly in always (or nearly always) showing a further peculiarity. This consists in a sort of parting between the middle and ring fingers, so that when the hand is laid on a flat surface the index and medius curve to the radial, and the ring and little finger to the ulnar side (figs. 14 and 15).

Many Mongol imbeciles also have a certain resemblance to cretins. This, however, is much more striking when they are

[1] John Thomson, *Edin. Med. Jour.*, June 1893.

older children or adolescents than in early infancy. In infancy they have really little in common beyond the mental backwardness (which is often not at all well marked in the Mongols), the frequently protruding tongue, the scanty dry hair, and the general fact that they are rather ugly-looking babies. On the other hand, the Mongol baby differs from the cretin in having a soft skin, rather slender small-boned limbs, a thin neck, with a normal thyroid and the peculiarity of the hands which I have already alluded to.

I should like especially to emphasise the fact that the characteristic shape of the hands in cretins is sometimes a very great help in diagnosing the condition, when (as in fig. 11) the face is less typical than usual.

I need not speak of thyroid treatment in cretinism and its effect on the prognosis, further than to say that while its careful administration from the earliest infancy may be expected to result in complete recovery so far as the bodily growth and deformity are concerned, the same cannot as yet be said with regard to the mental defect. The intellectual condition will be surely and steadily improved, but so far, I believe there has been no account published of any cretin who has quite reached normal intelligence.

6. *Eclamptic Imbecility*

The periodic occurrence of convulsions in infancy is often associated with the presence of mental impairment which may be the result of the fits, but much more frequently is merely another manifestation of the organic brain-disease which is causing them. All varieties of imbeciles, except cretins, are much more apt to suffer from fits than children of normal intellect.

The convulsions which occur in imbecile children may be of the regular epileptiform type. Very frequently, however, they consist, to begin with, merely in a sudden start—the head, arms and legs being suddenly jerked forwards. There is also a temporary loss of consciousness, and perhaps a little stertorous breathing ; and the child usually cries after the fit is over, as if it had made him uneasy.

Even where the fits are certainly the result of a damaged brain, it is very important to diminish their force and frequency if possible, because their recurrence is always detrimental to

intellectual progress. Sometimes a little can be done in this way by the use of sedatives, and especially by tonics and change of air.

There can, I think, be no doubt whatever that the occurrence of severe convulsions in healthy infants occasionally leaves a permanent mental defect, although generally it has no effect of this sort at all.

What I wish, however, specially to refer to here, is the fact that, after severe convulsive attacks, we may have a condition of prolonged stupor or temporary dementia, which, after the fits cease, gradually but completely disappears. I have seen three or four such cases. The following is the most illustrative of them:—

Case

On February 6th, 1891, a baby of six months (John S.) was brought to see me at the Dispensary on account of fits, which had been going on for four weeks. His parents were healthy, as were also their four other children; they had lost one child from "convulsive fits."

The patient was said to have been a perfectly healthy, bright, laughing child until four weeks before I saw him, when, without any known cause, he began to have a series of fits. In these he became unconscious, turned up his eyes, drew himself together, clenching his fists and flexing his arms and legs to their full extent. The "fit" lasted a very short time, but was rapidly succeeded by others exactly similar, until he had had from twelve to sixteen of them. Then there would be a pause for three or four hours, then another series of twelve to sixteen fits, and so on. According to the mother's account, this sort of thing had gone on for four weeks without a longer interval than four hours. Between the fits the infant was very drowsy.

On examination, the child was found to be fat and rather flabby, and to present signs of rickets about the wrists and some beading of the ribs. His head was well ossified and normal in shape, and the abdominal and thoracic organs appeared normal. There was no source of peripheral irritation to be found anywhere. His mental condition seemed to be very much changed from its previous normal condition. His eyes were very dull and heavy. He was unable to hold himself up at all. He could never be got to laugh, nor to look

at anything, however bright, nor to grasp any object put into his hand. He was altogether drowsy and apathetic, and had the appearance, indeed (fig 16), of a hopeless idiot.

Treatment.—He was given tolerably large doses of bromide and chloral thrice daily.

Progress.—The fits gradually became fewer and less severe, but there was little change in his mental state for more than a fortnight. On February 27th, however, he appeared distinctly brighter. He sat up and looked round him, and when a finger was put in his grasp, he held it.

By March 13th, the fits had ceased altogether, and although he still took the bromide and chloral, he continued to grow brighter and more intelligent, until, in April, his mother said that he was quite like himself again.

FIG. 16.—PROLONGED STUPOR FOLLOWING ECLAMPTIC ATTACKS.

There was no return of the fits, and the last time I saw him he was four years old, and seemed a perfectly normal child in every respect.

Meeting V.—February 2, 1898

DR JOSEPH BELL, *Ex-President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Dr Allan Jamieson* exhibited—

(*a*) CASE OF FAVUS, cured. Janet D., 16. Native of Edinburgh. Her mother died when she was six, and the family were somewhat neglected, but she only knows that she has had the disease for ten years. Commencing on the crown, it had pretty

nearly spread all over the scalp when she was admitted into the Royal Infirmary to Ward 38 on 15th June 1897. The hair was cut short, the scalp poulticed with boric starch poultices till quite clean. Pirogoff had stated that the disease could be cured if an ointment, having the following composition were applied, spread thickly on linen, and renewed till a scaly crust had formed. The disease in this way could be cured, he said, in course of several weeks. The ointment consisted of sulphuris præcip., 15.0; potassæ carbonat., 4.0; tinct. iodi. and Picis liq. ana., 50.0; adipis, 100.0. Lanoline was substituted for lard, and the ointment used after Pirogoff's directions for more than a month without the least effect. Systematic epilation was resorted to on July 20th; the head was washed daily with superfatted potash soap and warm water, and Pirogoff's ointment thoroughly rubbed in. On December 6th she was discharged as the head then seemed healthy. The hair was allowed to grow, but was still washed with the same soap, and a little of the salve rubbed in. The hair has in most parts grown well, though there are some thin areas, and there is no trace of disease. To all appearance she seems cured.

(*b*) CASE OF SYPHILIS, with peculiar pigmentary developments. The patient is a dressmaker, a native of Orkney, aged 39. There is no evidence available as to how the complaint was acquired. Admitted to Ward 38 on 8th January 1898. She stated that four months previously a rash appeared, somewhat like nettle rash on her arms, outer aspects of lower limbs, and back. This gradually extended in area, two months since affected the face and chest, and assumed a duskier colour. When admitted there was a dusky red macular, nodular, and papular eruption thickly scattered over most of the body. It scaled profusely on the face, back, and indeed wherever it was exposed to friction. On the forehead and face there were some brownish red nodules amid the macules. On the back and hands there were rings of minute papules apparently seated at sebaceous gland orifices or round hairs, inclosing patches of brownish or almost blackish pigmentation. The lower down the back the darker the hue, and it was very dark on the outer side of the thighs. On the chest the eruption was more scanty and less pigmented. The feet were free, the palms showed one or two spots. The throat was congested and there were some patches on the tongue. The cervical glands were slightly, the

inguinal glands markedly enlarged. Her general health was good, and there was no great anæmia. She was ordered a mixture containing one sixteenth of a grain of perchloride of mercury and two and a half grains of iodide of potassium twice a day. This was continued for a week, when pretty severe diarrhœa, not traceable to diet or exposure, set in. On the cessation of this under astringents and milk diet, mercurial inunction was substituted for the mercury by the mouth, and this has been continued till now. There was some evening rise of temperature with headache for three weeks, but these symptoms have now disappeared. The eruption too has steadily faded, till now all the nodules have vanished, and even the pigmentation is lessening. The papules have in most places been replaced by pale pigmentary dots, which can clearly be seen to be circumfollicular. There is a peculiar glossy appearance on the arms, and the characters now shown are not unlike a fading lichen planus. As to the cause of the intense pigmentation in this case, the patient informed me that her skin is one apt to show marks of bruising on slight injuries, such as trivial knocks or pinches, hence a soft non-resistant integument. She has also been in the habit of taking no fresh green vegetables, though as she partook of milk in considerable quantities, the tendency to the occurrence of scorbutus from this dietetic error was lessened.

2. *Dr Norman Walker* exhibited—

(*a*) A well marked case of LICHEN PLANUS in a young woman. The special characteristics of the disease were very well shown.

(*b*) A case of an unusual TERTIARY SYPHILIDE of a papilomatous type. The patient had been under treatment off and on for fourteen or fifteen years with moderate doses of iodide of potassium. Under large doses of iodide combined with the local application of Unna's mercurial plaster the case rapidly improved, and after four months' treatment was nearly well.

II. Exhibition of Specimens

1. *Dr G. A. Gibson* exhibited the heart of a patient who had died in his ward from the effects of combined mitral and tricuspid obstruction and incompetence. He had not seen

the patient during life, as she entered the infirmary late one afternoon, in a moribund condition, and died early next morning. At the necroscopy it was found that the mitral orifice presented the button-hole form of constriction, while the tricuspid showed union of the cusps with vegetations on them. The walls of the left ventricle were somewhat atrophied and the left cavities not enlarged. The wall of the right ventricle was greatly hypertrophied and the right cavities—especially the auricle—enormously dilated. The heart weighed 21 oz.

2. *Dr Harvey Littlejohn* exhibited—

(*a*) The CERVICAL VERTEBRAE from a man with dislocation between the third and fourth and fracture of the transverse process of the third vertebra. The posterior ligaments were torn but the anterior longitudinal ligament was uninjured. The injury was caused by a package weighing 1 cwt. falling off a barrow, which was being drawn by the deceased, on to his head and neck. The immediate cause of death was pressure on the cord from effused blood. Death took place within a few minutes.

(*b*) The ŒSOPHAGUS AND STOMACH from a case of carbolic acid poisoning presenting the appearances characteristic of a strong solution of the crystalized acid.

The stomach was strongly contracted in an hour glass form, the whole of the mucous membrane being corroded and transformed into the white pretty like material usually found in such cases.

(*c*) The LARYNX AND CERVICAL VERTEBRAE from a case of suicidal cut-throat. The wound of the skin was peculiar in as much as it proceeded transversely across the middle line of the neck for $2\frac{1}{2}$ inches, and then descended at right angles for 2 inches upon the right side of the neck. The edges of the wound were very irregular. Internally the epiglottis was cut through near its base, there were two cuts through the right side of the thyroid cartilage each half an inch long—the right superior cornua was completely separated together with a portion of the body of the thyroid, while the right arytenoid cartilage was also separated and lying loose in the larynx.

In the inter-vertebral disk between the fourth and fifth vertebrae there was a wound $1\frac{1}{4}$ inches long which penetrated

to the dura mater of the cord. The only large vessel divided was the right jugular vein.

3. *Dr W. Russell* exhibited (*a*) the HEART FROM AN UNUSUALLY RAPID CASE OF ENDOCARDITIS. The patient was a boy of 18 years, who had been at work on Wednesday as usual. He took supper before going to bed, and made no complaint of indisposition. His father, with whom he slept, was awakened about three in the morning by his son moaning and by the heat of his body. He tried to rouse him, but failed. He was seen on Thursday by an official from one of the Dispensaries. He, however, remained unconscious, and was taken to the Infirmary on Friday, where he died in the waiting-room before he could be taken to the ward. At the *post-mortem* examination there were recent vegetations on the aortic cusps ; there was a large area of embolic softening becoming purulent in the right cerebral hemisphere, and septic emboli in the kidneys and spleen. The vegetations showed multitudes of cocci which stained by Grana's method. The emboli in the vessels of the kidneys and spleen also contained cocci. The case was of importance because of the failure to discover any lesion to which the infective endocarditis might be attributed ; the very sudden onset of the symptoms, and the rapidly fatal course — death having occurred about sixty hours after the appearance of symptoms.

(*b*) A STOMACH WHICH HAD BEEN OPERATED ON FOR PERFORATION OF A GASTRIC ULCER. The woman had been in the Infirmary for some time with ill-defined abdominal symptoms, until one morning on his arrival he found that she had developed symptoms of abdominal perforation. Professor Annandale saw her almost immediately, and she was operated on in the side-room of the ward within four hours of the perforation having occurred. She died within two days after the operation. Owing to the perforation being on the lesser curvature near the cardiac opening, and being placed high up under the ribs and liver, there was some difficulty in stitching the perforation. The *post-mortem* showed that the perforation had occurred at the anterior edge of a large chronic ulcer which extended behind the upper margin of the stomach, and had for its floor the pancreas, to which it was firmly adherent all round.

The question that the case suggested to him was, whether it was not desirable, in cases where symptoms pointed to chronic ulceration, to enlarge the perforation, so as to enable the condition of the stomach in its vicinity to be determined and dealt with accordingly.

4. *Mr J. M. Cotterill* exhibited (*a*) A LARGE OXALATE OF LIME CALCULUS removed by the suprapubic operation from a young man of 24. The patient had suffered from cystitis for some weeks, but, with the exception of some perineal pain, had not presented the usual symptoms of calculus, such as hæmaturia, pain at the point of the penis, &c.

As his symptoms did not respond to the ordinary treatment for cystitis, Mr Cotterill sounded him, and finding a large and hard stone, performed suprapubic lithotomy. The stone was round, about the size of a golf ball, and covered by hobnail excrescences on the surface. It is interesting to note that such a stone failed to give rise to the usual symptoms. The patient made an uninterrupted recovery.

(*b*) A MECKEL'S DIVERTICULUM which he had removed from a young man of 18 who had been admitted to his wards a few days previously.

The patient, a strong, healthy-looking farm labourer, had been seized four days previous to operation with a sudden and severe pain in the lower part of the abdomen. This was followed by complete obstruction of the bowels; and vomiting commenced on the third day of the illness.

Mr Cotterill, having made the diagnosis of obstruction by a band, operated soon after the patient's admission. By this time there was fœcal vomiting and considerable collapse. The obstruction was found to be due to a Meckel's diverticulum about 8 inches long, springing from the ileum at a point unusually far above the ileocœcal valve, viz., 4 feet and 4 inches. (The large majority of these diverticula are found within a foot and a half of the ileocœcal valve). The diverticulum was attached to the umbilicus, and in the loop thus formed a coil of small intestine some 3 feet in length had been caught. The gut was dark brown but shiny, and was consequently released and returned, the patient's condition of collapse forbidding any more sweeping measures.

III. Original Communications

LOCALISATION OF FOREIGN BODIES BY X RAYS

By Dawson Turner, M.D., F.R.C.P.Ed., Lecturer on Physics, Surgeon's Hall; Assistant Physician-Electrician, Royal Infirmary, Edinburgh

I have quite recently had the pleasure of seeing Dr Mackenzie Davidson's method of localising foreign bodies by Röntgen rays. This method is a very complete and useful one, and it requires no acquaintance with geometry or mathematics. Dr M. Davidson takes two photographs on to one plate, and by reproducing mechanically, by means of silk threads, the course of the rays, he is enabled to exactly localise in the three dimensions of space, the position of the foreign body. Some further details of this method were now given. The only objection to this process is that it is lengthy and that the apparatus is expensive—a set costing about £10, 10s.

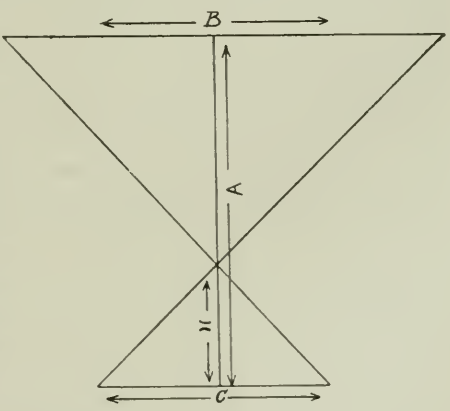
There is, however, a much shorter method of determining the exact depth at which a foreign body may lie, which does not involve the use of the somewhat complicated apparatus of Dr M. Davidson.

Two photographs of the foreign body are taken, and three distances are measured, viz. :—

(*a*) From the tube to the plate.

(*b*) Between the two positions of the tube from which the two photographs are taken.

(*c*) The distance between the two shadows of the foreign body on the negative.



From these three measurements the exact depth of the body can be at once calculated by using the following equation which depends upon Euclid's proposition that equiangular triangles are similar.

$$x = \frac{a \times c}{b + c}.$$

These three distances, (a), (b), and (c), are easily measured, and (x), or the distance of the foreign body from the skin or plate, readily calculated. Probably a third photograph would also be required, to locate the exact position of the foreign body in the other two dimensions of space ; and this photograph should be the first one taken, as my method would not give the depth accurately, unless the tube were placed in the perpendicular plane of the foreign body.

I now show a photograph of a potato in which a needle has been embedded at an unknown depth. The distance (a) was 33 cm., (b) was 10 cm. (c) is, say, 1 cm. Applying the formula

$$x = \frac{33 \times 1}{10 + 1} = 3 \text{ cm.},$$

we see that the needle is at a distance of 3 cm. from the under surface of the potato. I have received valuable assistance from Mr Shelford Bidwell in determining this formula.

An Adjustable Focus Tube

When Mr E. Swinton introduced last spring a tube with an adjustable anode, I at once saw that the tube could be improved in two ways—(1) Make the cathode adjustable instead of the anode, and thus get a fixed point for the point of origin of the radiation. (2) Cause the adjustment to be made by means of a magnet, so that the distance between the electrodes can be varied while a photograph is being taken. Mr Swinton's method was to incline the tube so as to cause the electrode to be moved by gravity. Mr Swinton also noticed, and at about the same time as the writer, the advantage of making the cathode movable instead of the anode. With such a tube, the resistance can be varied while a photograph is being taken. Experiments were shown to demonstrate the emission of X rays when the electrodes are at a proper distance apart, and the non-emission when they are at unsuitable distances.

Meeting VI.—March 2, 1898

Dr James Carmichael, *Vice-President, in the Chair*

I. Election of Member

Thomas Jackson Thyne, M.B., F.R.C.P.Ed., 2 Dean Terrace, was elected an Ordinary Member of the Society.

II. Exhibition of Patients

1. *Dr G. A. Gibson* and *Mr J. W. B. Hodsdon* exhibited a patient, aged 21, who had been successfully operated on for RUPTURED GASTRIC ULCER thirty hours after the perforation had occurred.

2. *Dr Norman Walker* exhibited a case of LICHEN SCROFULOSORUM, of which the following is a description :—

There appears to be pretty universal accord that the name of this disease is unfortunately chosen. The older dermatologists applied the term of lichen to any papular affection, but more modern teaching restricts it very much in its application, and all are agreed that this disease does not come into the restricted list. The alternative names suggested are Folliculitis scrophulosorum (Unna), Acne cachecticorum (Kaposi), and Scrophuloderma papulosum (H. Hebra). The disease is rare, Neumann giving it as 3 per 1000 in adults, and 5 per 1000 in children. It is, however, rather commoner than is usually supposed, on account of slighter cases being overlooked. This is illustrated very well by Crocker's statistics, which show that at the University College, where his cases are exclusively dermatological, the figures are 1·4 per 1000, whereas in his East London clinic in 1000 cases of skin disease out of 6500 general diseases the figures are rather more numerous than Neumann's. The disease has been investigated by several, of whom H. Hebra, Sack, and Jacobi regard it as a form of true tuberculosis of the skin, Jacobi having once found a bacillus which stained like the tubercle bacillus. Most observers consider that the lesions are probably produced by the toxins of the tubercle bacillus, and there are strong arguments at least in favour of this as against the other view. Ninety per cent. of the cases show evidence of tuberculosis, usually of the glands; phthisis is comparatively rare, but there is often a history of it in the family. Another argument

in favour of the disease being only indirectly tubercular, is its course. Almost all the cases get well even if left alone, which, unfortunately, is far from being the case in true tuberculosis.

The descriptions of the occurrence of the disease are very much at variance. While Hebra and M'Call Anderson describe it as a disease affecting young adults, the general experience in this country is that it is much commoner in children; all Crocker's cases occurred in children.

The disease is usually confined to the trunk, and consists of a number of papules which vary in colour from that of the skin, through yellowish red, up to brownish red. Their size varies, according to different observers, from a pin-head to the size of a lentil. They are either conical or flat, they have on their apices a small scale, or occasionally a pustule, and they tend to be arranged in circles or segments of circles—the natural arrangement of the hair follicles. Gradually the colour of the papules fades, and they ultimately disappear.

Under the microscope, the sections are found to consist of numerous cells, among which giant cells, epithelioid cells, and round cells are to be distinguished. The giant cell, which is always suspicious, is however by no means infrequently found in inflammation of the follicles. As already noted, only once has a somewhat questionable bacillus been found, and inoculation experiments have not proved successful.

J. I., aged three and a half, is the first case of this disease which has come under my notice. The boy was sent to me by Dr John Thomson, with a note to the effect that this was something with which he was not familiar, and a request for a diagnosis. Although in many ways it differed from the descriptions found in most of the books, I had little difficulty in recognising it from them. The distribution is exceptionally wide. No part of the body escapes, and on the limbs, as is seen in the illustration, the disease is widespread. It also occurs on the face, which is quite exceptional. The spots are only here and there arranged in circles, and so far as one case is evidence it seems to me to show that the disease is by no means necessarily limited to the follicles. This has already been noted by Sack, who found that one of the nodules which he examined microscopically did not correspond to a follicle. The lesions on the face and leg differed somewhat from the rest, showing a considerable halo, often of a peculiar livid

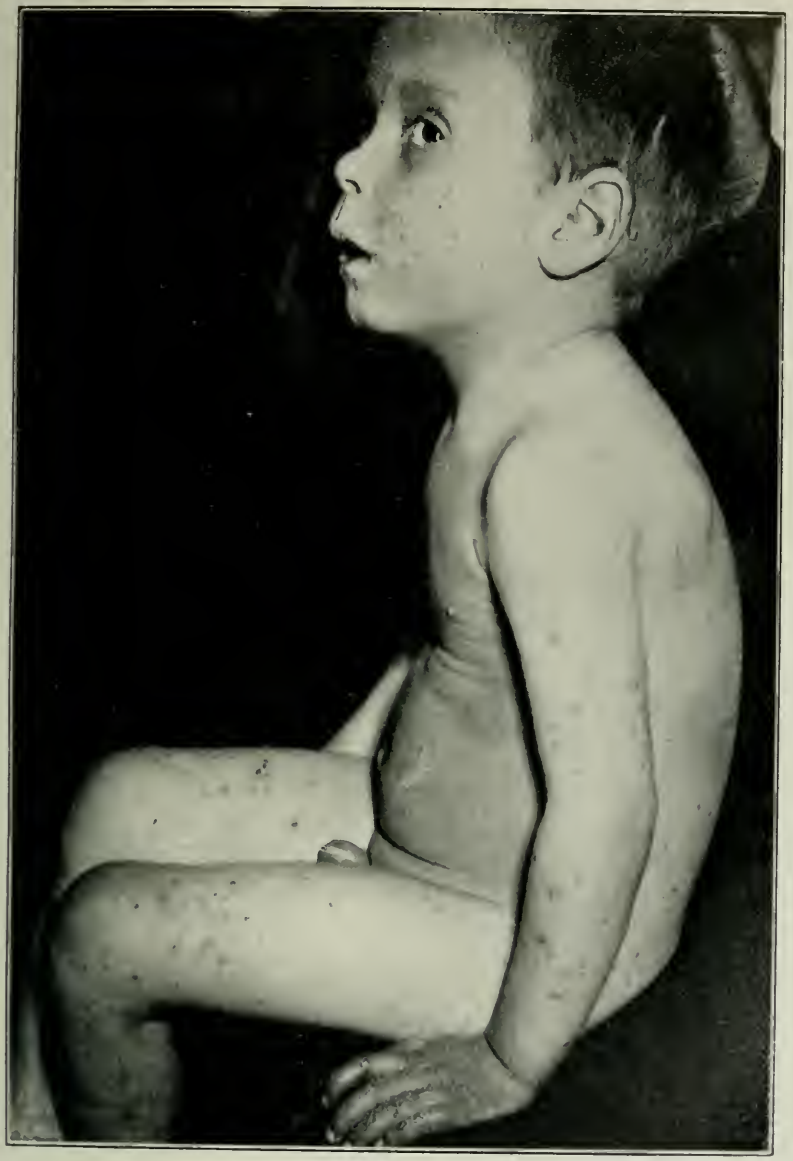


PLATE VI.—DR NORMAN WALKER'S CASE OF LICHEN SCROFULOSORUM.

bluish colour, which is also very evident in Bazin's disease, and must be familiar to many in connection with tubercular affections. This tendency of the spots on the legs and the face to differ from the others is noted by Unna. Some of the papules are covered with a yellowish scale, quite a number of them have a pustule at their apex, and some have the smooth top which undoubtedly recalls the lichen papule. The patient is the third in a family of four. His father and mother are both alive and healthy, and there is no history of phthisis on either side. Up to three years of age the boy was perfectly healthy, then he began to be listless and disinclined for play. About two months ago the eruption appeared. The spots first appeared on the cheeks, then on the limbs, the trunk being affected last. He takes his food well and sleeps well. After a brief examination, however, it seemed to me that there was some prominence of the abdomen, and a possibility of tuberculosis there. I sent the patient back to Dr Thomson with a letter indicating my diagnosis, and asking his opinion as to the possibility of abdominal tuberculosis. In conversation, he told me he was much interested in the diagnosis, as he had told his students that he had only twice before seen an eruption resembling this, and that in both instances it was in children dying of advanced tuberculosis. He was, however, unable to satisfy himself of the evidence of any abdominal tuberculosis in this case.

I have followed the treatment recommended by Hebra, namely, cod-liver oil internally and externally. It is not pleasant, but it appears to have been invariably efficacious, and under it the boy is certainly improving.

There are no illustrations of this disease, at least described as such, in any of the ordinary available atlases, but—and in this Dr Allan Jamieson agrees with me—there is in the Sydenham Society's Atlas, described under the name of Molluscum contagiosum, a picture of an arm which is at least very much more like Lichen scrophulosorum than the disease it purports to portray.

3. *Professor Annandale* exhibited a man who, nine months previously, had sustained a dislocation *backwards* of the head of the humerus, which had remained unreduced. The arm was very useless, and an operation was performed with the view of restoring mobility to the joint. The affected parts

were exposed by an incision carried along, and a little below the outer third of the clavicle and acromion process, a second incision being made along the inner border of the deltoid muscle. In the first incision, the attachments of the deltoid muscle were divided, and thus allowed this muscle to be turned outwards and downwards, and the head of the humerus and glenoid cavity to be thoroughly exposed. As the bone could not be replaced satisfactorily owing to the complication of a fracture of the glenoid cavity, its head was excised, and the attachments of the deltoid brought into position and stitched with catgut. Good mobility had resulted. Another advantage was claimed for this incision, namely, that it did not interfere with the principal vessels and nerves entering this muscle.

4. *Dr Byrom Bramwell* exhibited—

(*a*) A CASE OF PROGRESSIVE MUSCULAR ATROPHY, IN WHICH THE FACIAL AND OCULAR MUSCLES WERE AFFECTED.

The patient, a single woman, aged 64, was admitted to the Infirmary on 3rd November 1897, complaining of weakness in the arms and hands, difficulty in swallowing, and difficulty in articulation.

On examination, the case was found to be a typical one of progressive muscular atrophy of the Aran-Duchenne type. The duration of the disease was one and a half years. The small muscles of the hands, the muscles of the forearms, and the deltoids were extremely atrophied; the muscles of the upper arms markedly affected, the muscles of right arm being more atrophied than the left. The atrophy had commenced in the small muscles of the right hand. Some fibrillary twitchings were present.

Both sides of the face, the upper as well as the lower facial muscles, were markedly involved, the muscles of the right side of the face being more affected than those of the left. The patient was unable to close the right eye completely, owing to weakness of the orbicularis palpebrarum. She could not whistle or raise the upper lip to anything like the normal degree. The external muscles of the right eyeball were markedly involved, all the movements except the downward movement in the right eyeball being almost lost. There was also defective movement to the left in the left eye, due to paralysis of the left external rectus muscle. Both sides of the tongue, but particularly the right side, were atrophied. The right side of the tongue had a slightly uneven (hob-nailed)

appearance. Fibrillary twitchings frequently occurred in the muscles of the tongue. The uvula was somewhat œdematous. The reflex movements of the palate were not impaired.

The muscles of the lower extremity, particularly of the left thigh, were slightly affected. The left knee-jerk was absent, the right diminished.

There were no sensory disturbances, and no affection of the bladder or rectum.

During her stay in hospital, the patient was treated with hypodermic injections of strychnine. The dose was at first two minims three times a day, gradually increased until finally the patient was taking eleven minims of the liquor strychniæ three times daily.

Dr Bramwell remarked that in another case of progressive muscular atrophy which was in the hospital at the same time, the patient was able to take fourteen minims of the liquor strychniæ three times daily, hypodermically, without any of the ordinary toxic symptoms of strychnine poisoning being produced. These cases, and some others which he had observed, seemed to show that in progressive muscular atrophy there was an extraordinary intolerance to strychnine, fourteen minims three times a day, hypodermically, being for most persons a toxic dose. He was unable to say that in this or the other cases, in which he had tried the hypodermic injection of strychnine, had any marked benefit resulted from the treatment. The general condition of the patients had certainly improved, but the muscular atrophy had remained stationary or had increased in degree, notwithstanding that the remedy had been continued for a considerable period of time.

(*b*) A CASE OF TABETIC CLUB-FOOT (CHARCOT'S JOINT DISEASE) IN A CASE OF LOCOMOTOR ATAXIA.

The patient, a single man, aged 32, was admitted to the Infirmary on 15th December 1897, suffering from all the characteristic symptoms of locomotor ataxia.

During infancy the patient had suffered from polio-myelitis anterior acuta. The left leg, which was alone affected, was smaller and shorter than the right.

In October 1890 the patient contracted syphilis, a hard chancre, followed by sore throat, loss of hair and skin eruption, inflammation of the right eye and ptosis of the right upper lid. He was treated for four months, being at that time a soldier in India. While in India he had several attacks of malarial fever.

In May 1893, while jumping off a fence on to grass, he gave his right foot a jerk. The foot felt a little sore at the time, but he was able to walk on immediately. Two days afterwards, he was unable to get on his boot owing to swelling of the right foot and leg. The swelling extended half way up to the knee. The swollen parts felt soft and were red. The swelling was quite painless. The patient thought that the swelling was due to a suppurating corn which was at that time on the right foot. He remained in bed for six weeks. After two days' rest in bed the swelling in the leg disappeared, but the swelling in the foot remained. The swelling of the foot has continued since, and has gradually become harder and firmer. For the last four and a half years the condition of the foot has remained *in statu quo*.

It is interesting to note that none of the ordinary symptoms of locomotor ataxia were experienced until eleven months after the accident. Eleven months after the tabetic club-foot developed he began to suffer from lightning pains in the lower extremities. The symptoms were followed by the development of a girdle sensation, unsteadiness in walking, bladder symptoms, and loss of sensibility over the lower extremities, trunk, and arms, the head and neck being unaffected.

The patient is now unable to walk without assistance, the ataxia being very marked. The knee-jerks are absent. The Argyll-Robertson condition of the pupil is present. There is no optic atrophy. Sexual power is completely lost.

The thickness and swelling of the foot seem confined to the bones of the foot in front of the os calcis and metatarsus ; the metacarpal bones do not appear to be affected. The swelling is very firm and hard, irregular, and slightly nodulated. An inch and a quarter in front of the internal maleolus the right foot measures 1½ inches more than the left, being 11 inches in circumference. Occasionally, grating can be felt when the foot is moved on the leg at the ankle joint, but the ankle joint seems very slightly affected.

Dr Bramwell stated that this was the most marked case of tabetic club-foot which had come under his observation. The condition was rare. The case was extremely interesting, since the bone lesion was the first definite symptom indicative of locomotor ataxia which was developed. The lightning pains and ataxia were not noticed till eleven months after the develop-

ment of the bone disease. Dr Bramwell had seen other cases in which the joint affection had apparently developed as the result of a trivial injury.

Dr Dawson Turner had kindly taken a skiagraph of the foot, but the result was not very satisfactory, owing to the difficulty that the patient had in keeping the foot absolutely quiet for the time necessary to take a satisfactory photograph.

(*c*) A CASE OF ANEURISM OF THE THORACIC AORTA, IN WHICH A LARGE PULSATING SWELLING WAS PRESENT IN THE REGION OF THE RIGHT BREAST.

The case was peculiar, inasmuch as the aneurism had eroded the chest wall at a point much further to the right than is usually seen. Dr Bramwell had only seen one other case in which the aneurism had pointed so far to the right side.

The patient, a man aged 48, was admitted into the Infirmary on 1st February 1898. He was a labourer and had enjoyed good health until three years and eight months previously, then, while lifting a heavy bag of cement, he felt a sharp stinging pain across the breast; the pain extended from nipple to nipple. At the time of the accident, the patient felt giddy; but he had no sensation of anything giving way in the chest. The pain was accompanied with vomiting and purging, and was followed in the course of a few hours by swelling of the feet and scrotum, and in shortness of breath. He remained in bed for three weeks, and was unable to resume work for six or seven months after the accident on account of pain and shortness of breath on exertion. Prior to the strain he had been perfectly well and had been working hard as a labourer.

On returning to work, the shortness of breath and pain were so much aggravated that in the course of a few days he had again to leave off work. He remained off work for two months, then worked for three or four months. At the end of this time, the pain became so much worse that he had again to leave off work. He states that at this time there was no swelling, but that he could feel a small lump a little above the right nipple. He has not been able to work since, and has been more or less continuously under treatment in the Dundee Infirmary, the Edinburgh Infirmary, &c.

The external swelling is now about the size of an ordinary orange; it is the seat of a uniform expansive pulsation; all the physical signs of a large internal aneurism (marked dulness in

the course of the aorta, &c.), are present. A double aortic murmur is present at the base of the heart.

The patient states emphatically that he has never had syphilis. He had an attack of rheumatic fever twenty-four years ago.

5. *Mr Harold J. Stiles* exhibited (*a*) a child, æt. 1 year and 8 months, with a RIGHT-SIDED CONGENITAL INGUINAL HERNIA, in which the vermiform appendix could be distinctly felt by external manipulation through the scrotum. (*b*) A delicate child, æt. 1 year and 8 months, with an ordinary DOUBLE HYDROCELE, above which, on both sides, was what at first sight appeared to be a funicular hernia, but which was found on further examination to be a funicular hydrocele with a wide abdominal communication. The child's abdomen, which was very prominent, contained a considerable amount of fluid, and the case was no doubt one of tuberculous peritonitis, in which the fluid had gravitated into the patent funicular peritoneal processes. (*c*) A child, æt. $3\frac{1}{2}$ years, presenting all the typical features of CONGENITAL DISLOCATION OF THE LEFT HIP. As usual nothing abnormal was noticed until the child began to walk.

III. Exhibition of Specimens

1. *Professor Annandale* exhibited—

(*a*) A CONGENITAL HERNIÆ SAC containing a patent Meckel's diverticulum almost 5 inches in length. The patient was a boy, æt. 3, and as the hernia was irreducible owing to an adhesion of the diverticulum to the testicle, the sac, testicle, and diverticulum were removed, the end of the latter being carefully closed by means of a double row of lambdoid sutures about half an inch from the intestine. The patient had made a good recovery, notwithstanding an attack of scarlet fever which developed two days after the operation.

(*b*) AN APPENDIX which, together with the cœcum, was found adherent to the anterior surface of the right lobe of the liver. The case appeared to be one in which the embryonic position of the cœcum had remained persistent, and appendicitis having taken place, the adhesion to the liver resulted.

(*c*) A RUPTURED TUBAL PREGNANCY. The patient, who had been seen and her condition diagnosed by Drs Johnston and Kitchen, was brought to the Infirmary in a state of collapse, the abdomen being swollen and tender. The abdomen was at

once opened, when large quantities of extravasated fluid blood and clots escaped. The left tube, which was found to be dilated and ruptured, was removed together with the ovary—the pedicle being ligatured with strong silk. Protruding through the rupture the transparent membranes were seen, and floating in the waters was a small fœtus about half an inch in length. In addition to the ruptured tube and ovary, a large piece of omentum, which was infiltrated with blood clot, was ligatured and cut away. The peritoneal cavity was thoroughly irrigated with warm boracic lotion, and a glass drainage tube inserted. The recovery was rapid and good.

(*d*) A PEDUNCULATED PAPILLOMA OF THE BLADDER removed by suprapubic cystotomy.

(*e*) The parts from an old-standing injury of the elbow. The joint was firmly fixed in a useless position, and excision was performed. The preparation showed a fracture through the upper end of the radius, with displacement and a large mass of callus which had united this bone to the external condyle of the humerus.

(*f*) A number of seed-like bodies removed from the bursa under the anterior aspect of the deltoid muscle.

2. *Mr Alexis Thomson* exhibited the INTESTINES FROM A CASE OF INTESTINAL OBSTRUCTION in a newly-born infant, in which temporary relief had been afforded by enterostomy. The infant died a week after the operation, which was performed the third day after birth. *Post-mortem* it was found that the small intestine terminated in a large pouch situated in the left hypochondrium, and that the colon was represented by an independent tube, the size of an ordinary pencil, which terminated in a normal rectum.

Mr Thomson was indebted to Dr George Thyne for the opportunity of seeing the case, and for the specimen exhibited.

3. *Dr G. A. Gibson* exhibited—

(*a*) A DRAWING OF CHOROIDITIS FROM A CASE OF WAXY DISEASE OF THE LIVER, SPLEEN, AND KIDNEYS, of tubercular origin.

(*b*) DRAWINGS OF THE BRAIN AND MEDULLA FROM A CASE OF HÆMORRHAGE INTO THE VENTRICLES in a patient aged 23. The symptoms were paralysis of all four extremities, and external strabismus, with profound unconsciousness.

IV. Original Communication

LIVER CIRRHOSIS AND ITS VARIETIES

By Alexander James, M.D., F.R.C.P.Ed., Lecturer on the Practice of Medicine, Royal Colleges School of Medicine; Physician to the Royal Infirmary, Edinburgh

Somewhere in Book I. of his "Novum Organum" Bacon has said: "The greatest and, as it were, the radical difference between minds, as far as philosophy and the sciences are concerned, is this, that some are stronger and more fitted for marking the differences of things, others for noting their resemblances." Which of these peculiarities indicates the greater mind we need not pause to consider, but we may feel assured that both science and philosophy have been advanced as the result of the labours upon their problems of both these types of mind. I begin this paper by referring to this statement of Bacon, because in what I have to say to-night I have to ask you to seek with me for resemblances, rather than for differences. I am about to lay before you a set of propositions as the result of the consideration of which we may be able to trace a resemblance between processes ordinarily considered very different. To be brief, I wish to point out to you a unity as regards pathological processes between acute yellow atrophy of the liver and primary cancer of the liver. When I have said this you will probably be inclined to ask, What has this to do with the title of this paper, Liver Cirrhosis? To this I would answer, that in liver cirrhosis I would recognise the middle link in the connecting chain between those two pathological processes. Let us now, therefore, without more ado, proceed to the discussion of liver cirrhosis.

As you are all aware, two main forms of it have been described, the small, atrophic, or portal cirrhosis, and the large, hypertrophic, or biliary cirrhosis. In the atrophic cirrhosis it is believed that, as the result of some irritant, carried to the liver by the blood, specially of the portal vein, cell proliferation, and in time fibrosis, takes place around the portal radicals. The liver cells disappear, and the portal radicals are compressed, as the result of this fibrosis. As symptoms, we have therefore mainly those of portal obstruction—viz., gastro-intestinal catarrh, hæmatemesis, melæna, large spleen, and ascites; as

a rule no jaundice. If we get a *post-mortem* examination we find the liver small and misshapen, its surface rough, its edge nodulated and uneven.

In the hypertrophic cirrhosis, on the other hand, the view is that, as the result of catarrhal inflammation of the bile ducts, or of some other obstructing cause, there is not only re-absorption of the bile and jaundice, but cell proliferation round the ducts along to their terminations among the cells of the lobules. This cell proliferation in time becomes converted into fibrous tissue. As symptoms, we have, therefore, mainly chronic jaundice ; and if we get a *post-mortem* in such cases, we find the liver enlarged, firm in consistence, with its surface smooth, and with its normal, rather sharp edge well marked. Now the first point which I wish to draw attention to, is the transition forms between the hypertrophic and the atrophic cirrhosis. Those are extremely common, and I shall now lay before you a collection of such cases. They are a few collected out of many, and they have been specially selected to illustrate variations in the periods of time required for this transition.

Case I.—*Jaundice—Ascites—Enlarged Spleen—Hæmatemesis—Duration about Five Months*

R. J., aged forty-four, a commercial traveller in the spirit trade, with a good heredity, but a marked alcoholic history, was first seen July 22nd, 1896, suffering from jaundice.

He stated that he had always been a healthy man, although occasionally troubled with his digestion, till three weeks previously, when as the result, he thought, of a chill, his present attack of jaundice began. In spite of rest in bed, careful dieting, abstinence from alcohol, laxatives, etc., the jaundice had continued, and he was now suffering from severe pain over the region of the liver. On examination the liver was found markedly enlarged, and careful examination revealed the existence of a somewhat enlarged spleen, and of a slight amount of ascites. In spite of continued care as regards rest and dieting, his condition became worse, the ascites became more marked, swelling at the ankles made its appearance, and the superficial veins of the abdominal wall were very much distended. On August 4th, the ascites was so great that paracentesis was performed, and about 200 oz. of bile-stained fluid evacuated. After the tapping, the liver could be felt much enlarged, reaching down indeed nearly to the umbilicus. Its edge was sharp, and its surface felt even. The spleen was also enlarged, and could

be palpated distinctly in the left hypochondriac and lumbar regions.

After this tapping, the patient rapidly improved. The œdema of the legs quite disappeared, and the ascitic fluid did not collect again. He rapidly gained in strength, and although the liver and spleen still remained large, he went back to work. With his old work, his old habits, however, returned, and on November 12th he had severe and repeated hæmatemesis. At that date the liver was found to be distinctly smaller than before; its surface, however, was distinctly uneven. The spleen was also smaller than before, but it could still be easily felt in the left hypochondriac region. There was absolutely no jaundice. On November 18th — that is to say, about a week after the first hæmatemesis—he became comatose, and died. On *post-mortem* examination, the liver tissue showed a mixture of large and small cirrhosis.

Here, then, was a case in which the transition of a large or biliary cirrhosis into a small or portal one might be said to have occupied a period of only a few months.

Case II.—*Jaundice—disappearing after six months, followed, after three years, by Symptoms of Portal Obstruction—Ascites—Enlarged Spleen*

Elizabeth B., aged twenty-two, born and residing at Kirkliston, was admitted to Ward XXXIII., on May 24th, 1897, complaining of swelling in the abdomen and general weakness, and stating that she had been ill for over eight years. Her family history is fairly good, and she has always been comfortable at home. She has never worked, owing to her present illness. She has had three attacks of measles, and also scarlet fever and whooping-cough, when quite young.

Her present illness began eight years ago. She says that she got a chill, and felt so ill as to go to bed. After a few days she became jaundiced, and this attack of jaundice seems to have lasted for about six months. She says that the jaundice then disappeared, but that she has never felt strong. Ever since, she has felt more or less feeble and dull, and has been very liable to catch cold. About five years ago she noticed that her abdomen was becoming larger, and this increase in its size has gradually become more marked. For a time she felt pains in the upper part of the abdomen, but those have disappeared for the last year or two. During the last month, the abdomen has increased more rapidly in size, and she has been feeling specially feeble, so she sought admission to the Infirmary.

State on admission.—She is 5 ft. 2 in. in height, and weighs 6½ st. Her muscularity is poor, but the most obvious morbid appearance is the very much distended abdomen, with extreme

distension of the superficial veins, causing a marked "caput medusæ." Her pulse is somewhat rapid, about 116, and her temperature slightly subnormal. There is no jaundice.

On her admission, the abdominal distension caused so much distress that tapping was resolved upon. This was done, and 176 oz. of clear, slightly yellow fluid were drawn off, as the result of which the patient felt much easier.

Alimentary system.—The patient complains of dryness in the mouth on waking from sleep. The tongue is smooth and red, and exhibits deep transverse fissures. Her appetite is fairly good, but she complains, especially of late, of pain in the stomach after taking food, of acid eructations, and of vomiting after meals. She has never vomited blood. Bowels for years have tended to be loose, two or three motions every day, and the fæces are watery. After the abdominal tapping, the liver dulness was found to extend from the fourth rib above to an inch below the costal margin in the nipple line. In the middle line it extended an inch below the lower end of the sternum, and in that region the lower border of the liver could be palpated. Its surface could be felt to be uneven, and its edge rounded.

Hæmopoietic system.—On percussion the spleen was found to be distinctly enlarged. Its anterior border could be felt distinctly in the left hypochondriac and lumbar regions.

The circulatory system is normal, with the exception that the pulse is rather rapid, and that there are hæmic murmurs over the heart, and in the veins of the neck.

The respiratory system is normal, and so also is the integumentary system except that she has a little œdema of the feet and ankles.

Urinary system.—The urine is normal as regards quantity, is pale in colour, and has an acid reaction. Its specific gravity is usually low, but it contains no bile nor other abnormal constituents.

Reproductive system.—Here all that need be mentioned is that, although she is twenty-two, she has never menstruated.

During her stay in hospital she improved very much in condition. The tapping did not require to be repeated. The œdema of the feet disappeared, and the distension of the superficial abdominal veins became much less marked. On the 18th of June the patient became slightly jaundiced, but this completely disappeared after a few days. The diarrhœa became much less troublesome, and she was discharged greatly improved on the 12th of June. The medicinal treatment was mainly small doses of mercury internally.

In this case the symptoms of portal obstruction seem to have shown themselves some three years after the jaundice.

Case III.—*Jaundice of some Thirty Years' Duration—Enlarged Liver—Enlarged Spleen*

B. S., aged sixty, a weaver, was admitted to Ward XXX., August 26th, 1895, complaining of jaundice and lowness of spirits, and stating that he had been ill for thirty years. He has a good family history, and his surroundings at home are comfortable. He has not wrought for nine years, owing to impaired vision from double cataract. He tells us that a sister of his own has also been subject to jaundice for many years.

His illness began thirty years ago, by an attack of dyspepsia, during which he became jaundiced. This jaundice has remained ever since, sometimes better, at other times with a little dyspepsia, being worse. During the exacerbations he has noticed the urine darker in colour, and the fæces paler, and early in the course of the disease, he states that he was greatly troubled with boils on the skin. He has never complained of itching.

Present condition.—Patient is a fairly developed man, and is markedly jaundiced, the face and chest showing the colour best. His temperature is subnormal, pulse is usually about 70, and fairly strong and regular. The skin, especially about the epigastrium, feels greasy.

Alimentary system.—His appetite is fairly good, though his mouth is dry and sticky in the mornings. The tongue is large and flabby, and shows a yellowish fur. After taking food, he has a feeling of uneasiness in the stomach, and he is troubled with flatulence. At times, after food, he feels sick, and may vomit. The bowels tend to be constipated; fæces vary somewhat in colour—at present they are rather pale. On examination of the abdomen the lower border of the liver could be palpated in the epigastric region. It felt firm and the edge rather sharp. On percussion in the nipple line its area of dulness extended from the fourth rib above, to $1\frac{1}{2}$ inch below the costal margin.

Hæmopoietic system.—No enlarged glands were detectable, but the spleen was markedly enlarged. It could be felt in the left hypochondriac and lumbar regions, extending to about $3\frac{1}{2}$ inches from the umbilicus. By percussion in the axillary line its area of dulness was found to extend from the eighth rib above to within an inch of the iliac spine.

The circulatory and respiratory systems were normal.

Urinary system.—The urine was about 50 oz., of a dark amber colour; acid reaction and a specific gravity of 1020. With nitric acid, it gave traces of bile. Sugar and albumen were not present.

His integumentary system, with the exception of the jaundice, was normal, as also was his nervous system, with the

exception of the cataract, and the feelings of lowness of spirits, which, he says, become worse with the exacerbations of the jaundice.

He was treated by rest in bed, convalescent diet, and cold water enemata every morning, and for drugs he got 20 grs. of chloride of ammonium in infusion of gentian thrice daily. He rapidly improved, and on the 1st of September it is noted that both liver and spleen had much diminished in size. A few days afterwards he was discharged feeling fairly well.

In this case the hypertrophic or biliary cirrhosis was, I believe, the main condition, but the greatly enlarged spleen indicated the possibility of some obstruction to the portal veins. It was not, however, great, even although the jaundice had lasted for thirty years.

In the liver cirrhosis of children, mixed or transitional forms are the rule. Of such I shall quote two examples.

CASE IV.—James C., aged six years, from Cowdenbeath, was admitted to Ward XXX., on March 16th, 1895, complaining of swelling of, and pains in, the abdomen, and of occasional diarrhœa.

His illness dated from an attack of measles two years previously. This attack had left him decidedly weaker, and about eighteen months ago swelling of the abdomen was first noticed. This swelling became more and more marked, but no other symptom seems to have appeared till January 1895, when diarrhœa set in. The stools were noticed to have a greenish colour and a fœtid odour. About this time also, pains in the epigastrium were complained of. These got worse in time, causing him to cry out, and as they were continuing, he was recommended to come to the Infirmary.

On his admission he was seen to be fairly well grown for his age, but he was lacking in muscularity, and his thin arms and legs contrasted markedly with his large abdomen. The conjunctivæ showed a distinctly yellow tinge. Temperature normal, pulse usually about 90.

Alimentary system.—Tongue clean and pink in colour. He has a good appetite. He has occasionally some pain in the epigastric region, but this bears no relation to the taking of food. He has no vomiting, and the bowels are fairly regular. The fæces are fairly well coloured.

On examination the abdomen was found large and globular in shape, with the superficial veins markedly distended. At times a transverse ridging (Wyllie's "ladder pattern"), due to coils of distended small intestine, could be made out through the thin abdominal wall.

On palpation the liver was felt enlarged. Its lower border could be felt sharp and hard, and here and there over its edge

and anterior surface distinct nodulation could be felt. Its percussion dulness extended in the nipple line from the third rib above, to 1 inch below the costal margin.

The spleen was enlarged, and could be palpated in the left hypochondriac and left lumbar regions, and its percussion dulness extended over 6½ inches in its long diameter.

With the exception of a trace of bile in the urine, examination of the other systems revealed nothing abnormal. This boy remained in the Infirmary for some weeks, and was discharged *in statu quo.* On his returning home, he soon became worse, ascites supervened, and after tappings, recurred, and he died in the December following.

CASE V.—The next case is that of B. G., a school-girl, aged ten years, who was admitted to the Infirmary, January 18th, 1896, complaining of swelling of the abdomen, occasional pains in the right side, and troublesome cough and spit.

According to her mother's statement, she has been delicate ever since she had a fall down the stairs, when she was two years old. About a year after this, when she was three years old, she awoke one night with severe pain in the right side. Hot fomentations were applied, and the pain subsided, but in the morning it was noticed that she was jaundiced. The jaundice, however, does not seem to have been very bad, for on her being taken next morning to the Children's Hospital, she was treated as an out-patient. Although apparently not seriously ill, she has remained weakly. She has had cough in the winter months and frequent spitting of blood. Two years ago, as she was feeble and as the jaundice seemed rather worse, she was taken in to the Infirmary for a few weeks. She was discharged improved, but still feeble and slightly jaundiced. Last winter her cough was severe, and she brought up considerable quantities of blood. With the advent of summer, she improved, and was able to go to school. This winter she has been again complaining of pain in her side and cough, and so admittance to the Infirmary was asked.

State on admission.—She is a dark-haired, dark-eyed, rather gypsy-looking girl, thin and anæmic, with the skin dry and earthy-looking. Conjunctivæ slightly yellow. Pulse and temperature normal. Appetite variable, tongue clean, lips and gums well coloured. Bowels regular, fæces coloured.

The abdomen was markedly enlarged, and showed the superficial veins greatly distended. No distinct ascites. Liver greatly enlarged, its percussion dulness in the nipple line extending from the fourth rib above, to 2½ inches below the costal margin. Palpation showed it to be firm in consistence, and showed, also, little nodulations over its surface and at places on its otherwise sharp edge.

The spleen was much enlarged; it could be palpated in the left hypochondriac region, and its anterior border reached to within an inch of the umbilicus.

The urine showed a trace of bile, and signs of slight consolidation and catarrh could be made out at the right lung apex; the other systems presented nothing abnormal.

This girl, after six weeks' residence in the Infirmary, left somewhat improved in condition. I then lost sight of her, but I have been able to ascertain that she died with head symptoms and with indications of portal obstruction in July 1897.

Now in all those cases, we can, I think, recognise a transition, for we recognise conditions in which the symptoms and physical signs of biliary cirrhosis manifested themselves more or less markedly, and in which, after a time, one or other of the symptoms and signs of the portal cirrhosis made their appearance. In the cases which I have quoted, you will all have remarked great differences in the periods of time occupied by the transition. You will now ask, What is the cause of those time differences? Let me now try to answer this question.

The normal human liver is made up of liver parenchyma and a relatively small amount of interstitial or connective tissue. In normal conditions, we have to suppose that the nutritive power possessed by those two tissues is accurately balanced, so that, as time goes on, the parenchyma and interstitial tissue retain the same relationship to one another as regards amount. Suppose, now, that anything occurs to disturb this balance. It is evident that this disturbance can only be in the way of diminishing relatively the trophic power of the parenchyma, or of increasing relatively that of the interstitial tissue. Either way the result would be the same, viz., disappearance of parenchyma and excess of connective tissue. The difference in the times occupied in the development and spread of the cirrhosis will correspond with differences in the degree of strength of the factor which disturbs the balance. Thus, in the first case, in which the cirrhosis spread rapidly through the liver in the course of a few months, the factor was a very strong one, being simply the fact of his liver tissues having been soaked for years in alcohol. In the other cases the factors, whatever they were, have been correspondingly less potent.

Another question which may be asked here is why the liver should be large in biliary cirrhosis and small in portal cirrhosis. In a general way the explanation is, I believe, that in biliary cirrhosis it is only the biliary function of the liver cells which is interfered with, the numerous and complex other functions of the cells going on as before. In the so-called portal cirrhosis, on the other hand, the irritant, whatever it may be, is in the blood which supplies the liver cells. Consequently, we may suppose,

these liver cells will suffer as regards the performance of all their functions. *Ceteris paribus*, therefore, the disorganisation of liver cell function will be greater when the irritant or disturbing factor is in the blood, and so diminution in size of the liver will more readily occur.

I next quote a case of cirrhotic changes in the liver, limited, however, to the bile duct walls and structures around.

Case VI.—*Gastric Ulcer causing adhesion to Liver—Growth of fibrous tissue along Glisson's capsule. Jaundice—Ascites*

R. M., aged thirty-nine, a labourer, from Cardenden, was admitted to Ward XXXI., April 20th, 1897, complaining of jaundice and of a sharp pain in the region of the liver, coming on daily and lasting about an hour, also of discomfort in the stomach. He stated that he had been ill for three months.

His family history is fairly good, as also are his surroundings at home and at work. He admits, however, to have been in the habit of taking two or three glasses of whisky per day.

His present illness seems to have begun three months ago. He says that then he got a wetting, and soon afterwards he had to complain of pain over the liver. Shortly afterwards he noticed that he was becoming jaundiced, and this, with discomfort after food, and weakness continuing, caused him to seek admission to the Infirmary. The jaundice has persisted up to the present time, and he states that for some time previous to his admission, although the pain has not troubled him so much, he has experienced a heavy feeling after food, and he has been much troubled with flatulence. Occasionally, too, after food he suffers from a griping pain in the upper part of the abdomen, which shoots up between his shoulder-blades.

State on admission.—He is 5 feet $5\frac{1}{2}$ inches in height, and weighs 8 stone $8\frac{3}{4}$ lbs.; his weight previously having been 11 stone. His muscles are soft and flabby, and he is markedly jaundiced. His pulse-rate is about 60 per minute, and his temperature is rather subnormal.

Alimentary system.—He complains of a bad taste and occasional dryness of his mouth. His tongue is flabby and slightly furred. His appetite is fairly good, but he feels heavy and uncomfortable half an hour after meals, and suffers then from flatulence and acid eructations. The bowels tend to be constipated, and the fæces have, since his illness, for the most part been pale in colour. The area of liver dulness begins above at the fourth interspace in the nipple line, and extends to an inch and a half below the costal margin. In the nipple line, and in the epigastrium, the liver can be felt. Its surface is smooth, and its lower border is a little rounded.

PLATE VII.—TO ILLUSTRATE DR JAMES' PAPER.

LIVER IN CASE VI. (R. M.). THE THICKENED GALL-BLADDER IS SHOWN ABOVE, THE HORIZONTAL SECTION BELOW SHOWS THE FIBROUS TISSUE ROUND THE BILE DUCTS AND PORTAL VEIN AND THE CONTINUATION OF THIS THROUGH THE LIVER SUBSTANCE.

Hæmopoietic system.—The spleen appears on percussion to be of normal size, and there are no enlarged lymphatic glands. The circulatory and respiratory systems present nothing abnormal.

Integumentary system.—The jaundiced colour of skin is well marked all over, and the characteristic smooth, greasy feeling is present. He complains of itchiness of the skin, especially on the soles of the feet.

Urinary system.—The urine is normal in quantity and specific gravity, but is of a dark olive green colour, and gives markedly the bile pigment reaction.

In the belief that this was a case of catarrhal jaundice, running a very chronic course, the patient was treated by rest, careful dieting, and small doses of mercury. As improvement was slow, other means of treatment employed were chloride of ammonium, Durande's remedy of turpentine and ether, and cold water enemata. The patient, however, considered that the mercury had been of the most use to him, and so, after a week, it was again resorted to. As the time wore on, the patient expressed himself as feeling better, but the jaundice, though slightly better, still continued, and the liver increased in size, its lower border becoming easily palpable. Believing that a change home might do him good, he left the hospital on May 29th.

He remained at home till the 13th of July, when, finding his condition getting worse, he returned to hospital. His liver was found to be just as large as before, the percussion area of his spleen measured 6 inches in the long direction, and he had marked ascites. He was tapped on the following day, and 116 oz. of bile-stained fluid drawn off. After this he steadily got worse, began to vomit all his food, and got more and more emaciated. He died comatose on July 27th. *Post-mortem report.*—Body markedly jaundiced; considerably emaciated. *Abdomen.*—Pyloric end of stomach firmly adherent to under surface of liver. Stomach somewhat dilated. At about 1½ inch from pylorus towards its lower curvature was a healed gastric ulcer, with puckered cicatrix. The mucous coat at this point was completely destroyed, and there was extensive fibrous induration and thickening in the submucous and muscular coats for some distance around.

Liver.—Weight 4 lbs. 4 oz. Gall-bladder, about the size of a hen's egg, was distended with dark brown bile; its coat was considerably thickened, specially at its neck, where the thickness measured ¼ inch. Its mucous membrane showed a granular appearance, but was otherwise normal.

The portal fissure was the seat of very extensive fibrous growth, which on horizontal section appeared as an irregular area fully 1½ inch in diameter, around the bile ducts and portal vein. The two main bile ducts were considerably compressed,

and their walls thickened, but a fine probe could be passed along them. The common bile duct was normal, as was the opening into the duodenum. The compression, with resulting jaundice, had been on the portal fissure ; above that level many of the bile ducts showed considerable dilatation. There was also fibrous thickening in the portal spaces which extended down to the minute branches. The liver tissue was deeply bile-stained, of a dark green colour ; it also showed fatty infiltration, but there was no marked cirrhosis in the lobules ; there was chronic venous congestion.

Spleen.—Weight 5 oz. Showed chronic venous congestion, along with some thickening of the trabecular tissue.

Kidneys.—Each weighed 6 oz. Cortex slightly atrophied, but no interstitial growth. The other organs showed nothing noteworthy.

Here a gastric ulcer had cicatrized and become adherent to the under surface of the liver. From this as a source, the fibrosis had, as it were, like a new growth, spread through the liver along Glisson's capsule, involving mainly, however, the bile duct walls.

I shall now quote a somewhat similar case from Frerichs, in which the fibrosis from a gastric ulcer spread to the liver, but mainly involved the portal vein.

"*Diseases of the Liver.*" Frerichs. Vol. I. p. 252.—*Chronic Atrophy of the liver, with considerable enlargement of the branches of the portal vein.—A small ulcer at the pylorus, without any constriction at the opening.—Distinctly visible peristaltic movements of the stomach.—Death from exhaustion.*

"Adam Blaschefsky, a day-labourer, aged fifty-three, was admitted on the 21st November 1854.

"The patient was emaciated, but free from œdema, and from any abnormal colouring of the skin ; he had complained for a long time of pains in the epigastrium, and had often brought up the food he had swallowed, without the admixture of any foreign substances. His appetite was but slightly impaired, the stools were regular, of normal consistence, but pale. The thoracic organs were intact, the respiration free, the heart's action normal, pulse 62. The abdomen was collapsed, and the abdominal walls, when felt, appeared unusually thin. Upon close examination, the contour of the stomach, distended with gas, was distinctly visible ; on percussion the full tympanitic sound of the organ could be distinguished from the more abrupt sound of the small intestine. Half an inch to the right, and above the umbilicus, was observed a somewhat prominent tumour which felt hard and

uneven, was tender upon pressure, and could be shifted about. This was of course assumed to be a cancer of the pylorus. The hepatic dulness was reduced: in the median line it amounted to $\frac{4}{5}$ inch, in the mammary line $1\frac{1}{5}$ inch, and in the axillary line to 2 inches.

"Under the use of tincture of rhubarb, and extract of belladonna, the vomiting subsided, but the emaciation increased with tolerable rapidity, notwithstanding sufficient nourishment and an improved digestion. The tumour at the pylorus changed its position repeatedly; it was found sometimes to the right, and sometimes to the left, of the umbilicus, and at other times beneath it, right in front of the vertebral column. . . .

"On the 22nd of December the patient, who felt comparatively well, asked for his discharge, but so soon as the 27th he returned in a state of great exhaustion. Excesses in diet during the Christmas festivities had induced severe catarrh of the stomach. The tongue was thickly coated; there was great tenderness of the epigastrium and no appetite. Vomiting of mucous matter without any admixture of blood suddenly supervened. . . . The patient became rapidly collapsed, and died from exhaustion on 14th January.

"At the *post-mortem* examination the following were the most important changes found:—

"The lining membrane of the stomach was tumid, of a dark livid hue, and covered with a layer of mucus. The veins, especially those of the small curvature, were much enlarged and distended, and dark blue. Just before coming to the pylorus there was an ulcer, half an inch long, three lines deep, and equally broad, the margins of which were still covered with puckered mucous membrane, and which presented a slightly elevated edge formed of cancerous infiltration of the submucous areolar tissue.

"The liver exhibited all the characters of chronic atrophy in a marked degree. It was small, flabby, and tenacious; its outer surface was uneven, and covered with flattened prominences, about $\frac{1}{4}$ line in breadth. Its cut surface discharged an enormous quantity of thin fluid blood from numerous widely gaping mouths of vessels which were everywhere visible. The calibre of these vessels was considerably enlarged. . . . This enlargement extended throughout the entire subdivisions of the portal vein, as far as its capillary ramifications, and could also be observed in the hepatic veins, although to a less extent. The tunica adventitia of the portal veins was very considerably thickened. . . . In tracing the ramifications of the portal vein, several of its branches, and at the same time apparently also branches of the hepatic artery and of the hepatic duct, were found enclosed in a thick common sheath of

areolar tissue, which likewise contained a network of elastic fibres, each individual vessel being furnished in addition with a tunica adventitia of its own. . . . In thin sections of the dried hepatic tissue thick sheaths could be seen everywhere surrounding the apertures of the vessels, some of which were round, and others elongated; these sheaths occasionally enclosed branches of the hepatic artery, and contrasted remarkably with the thin-walled branches of the hepatic vein."

In this way the portal capillaries seem to have been obstructed, for "in many parts of the liver even water injected into the portal vein, could not be forced through them." Frerichs then goes on to say: "In this case the primary cause of the atrophy, of the destruction of the capillaries, and of the disappearance of the secreting cells, depended apparently upon the disease of Glisson's capsule, which, proceeding from the ulcerated spot at the pylorus, extended into the fissure of the liver, and along the course of the portal vein, as far as its ramifications. The disease attacked the portal vein itself, as proved by the marked structural changes in the walls of that vessel. . . . In the trunk and larger branches it gave rise to thickening, paralysis of the muscular tissue, and dilatation, but in the smaller branches, and in the capillaries, to the abnormal conditions just described, obliteration, etc."

Frerichs also quotes a case in which a diminution in the amount of liver parenchyma and increase in the fibrous tissue occurred as result of extension from old-standing peritonitic and perihepatitic adhesions. (Frerichs, *ibid.*, vol. ii. p. 101.)

In these two cases quoted in detail, the passage of the fibrosis from the gastric cicatrix along Glisson's capsule, indicated again a disturbance in the balance of parenchyma and connective tissue nutrition. Whether this was due to lowered parenchyma trophic power, or to increased connective tissue trophic power, need not yet detain us.

We have to notice, however, again the interesting phenomenon, in the fact that whilst in the bile duct fibrosis the liver was large, in the portal vein fibrosis the liver was small. We explain this on the ground previously stated, viz., that in the former case the biliary function alone would be interfered with. The other functions of the liver would go on, and though there was at times evidence of impairment of the portal circulation, this was not continuous. Moreover, the hepatic artery circulation would always go on. In the second case, on the other hand, the diminution in the amount of portal and hepatic artery blood would lead to diminished general function in the liver, and in time to diminution in structure.

As closely related to the last two cases, in which we recognised that a fibrotic process had spread into the liver from outside, let me now quote to you certain cases in which a

fibrotic change seems to have occurred in the liver as the result of a local disturbance of nutrition in the interior of the organ. You all know that when a patient appears with a distinctly recognisable tumour of his liver, you are very apt to diagnose a syphilitic gumma, if in time that tumour disappears, and the patient recovers more or less completely. I am persuaded, however, that localised swellings or tumours of the liver may occur independently of syphilis, and I shall now quote three cases, in which "probably malignant" tumour of the liver was diagnosed, and in all of which the subsequent course has demonstrated that the lesion has been neither malignant disease nor syphilis. Moreover, in at least two of these cases, it has been followed by cirrhotic changes in the organ.

Case VII.—*Enlarged Liver of over twenty-four years' duration—Supposed Tumour—Ascites—Enlarged Spleen, followed by large Cirrhosis*

W. L., aged forty-one, a joiner, residing in Edinburgh, was admitted on January 20th, 1874, into Ward II., old Edinburgh Royal Infirmary.

Complaints.—Patient complains of swelling of the abdomen, shortness of breath, slight pain over the region of the liver, and suffers likewise from general weakness.

History of present illness.—He states that previous to his present condition he had been exposed to cold and wet, and it is only three weeks ago that he began to feel great uneasiness about his belly. He soon noticed it to swell rapidly, to such an extent as to be obliged to give up work. He has since then lost his strength very much, and dyspnœa has greatly increased, in consequence of the swelling of the abdomen. His appetite has greatly failed since the onset of the present illness.

Previous health.—Had an attack of small-pox about twenty years ago. Four years since he suffered from rheumatic pains on his right shoulder and back. Long ago he used occasionally to perspire a great deal at night. During the last twelve months he has been complaining of great uneasiness about the stomach, after meals, chiefly after dinner and supper. He says it used to swell so much after taking food that he was obliged to go out for a walk, in which he found great relief.

Social conditions and habits.—Favourable; he states that beer was his favourite drink.

Family history.—His father died at the age of forty-one, of typhus fever; his mother and sisters are alive and healthy.

State on admission.—He is well developed and of average height; his muscles are flabby. He is able to walk about; in bed he lies easiest on his back. He has rather a sallow complexion

and anxious countenance. There is some œdema on the back —the legs are also œdematous. The abdomen is greatly distended. Temperature 98·6°, pulse 72 per minute.

Digestive system.—Tongue pale, appetite bad. With the exception of a sense of fulness in the stomach after meals, digestion is normal. No vomiting, bowels somewhat confined. Abdomen very much distended, its circumference in greatest diameter 38¾ inches. Percussion tympanitic, within a circle several inches wide of the umbilicus, dull all over the rest of the abdomen, absolutely so on the flanks. Dulness shifts on altering the position of the patient. Distinct fluctuation on palpation. The liver dulness begins above at the sixth rib and extends to the costal margin. Below this it cannot be distinguished from the fluid. Percussion of this region causes slight pain.

Urinary system.—Amount, 30 to 36 oz., specific gravity 1020, muddy yellowish colour, with a considerable heavy deposit, which consists of purple or pink urates—no albumen. By the microscope, oxalate of lime crystals are seen.

Thorax.—Respiratory system.—Respirations 20 per minute. No pain, but some dyspnœa. Examination reveals the presence of fluid at both bases, especially the right.

Circulatory system.—No palpitation nor pain at the precordia. The position and size of the heart are normal, and there are no murmurs. The veins of the abdominal wall are markedly distended.

Purgatives and diuretics were employed, but in spite of this, no diminution in the ascitic fluid occurred. Accordingly, on February 1st, 1874, the abdomen was tapped, and 360 oz. of fluid withdrawn. On examination of the abdomen after paracentesis, inspection revealed a rounded tumour of considerable prominence occupying the middle and right epigastric regions. The infra-costal margin on the left side was distinctly seen in outline, whilst that of the right side was obscured and obliterated by the tumour, which passed underneath it to blend with the liver. The tumour does not move up and down with the movements of the diaphragm.

Palpation.—On applying the fingers gently over the tumour it is found to have a more or less boggy feeling, and crepitates on pressure, this crepitation is more especially perceptible when the patient takes a forced inspiration (perihepatitic friction?). On exerting firmer pressure, the tumour feels dense and resisting, and seems to occupy the anterior surface of the liver, which can be felt on a deeper level, hard, flat, and resisting, and as low down in the abdomen as the umbilicus. The position of the tumour, visibly projecting in the epigastric region, measures 4 inches in the long diameter, which lies transversely, and 3 inches in the short diameter, which lies obliquely along the right infra-costal border. The sharp margin of the liver can be

distinctly made out sharp and hard, and on a level with the umbilicus in the mammillary line.

Percussion.—Liver dulness extends from the fourth intercostal space in the right mammillary line, down to $2\frac{1}{4}$ inches below the free margin of the ribs. Transversely it extends from the right hypochondrium across the epigastrium, where it begins to blend with the spleen dulness. The splenic dulness is increased, measuring vertically 6 inches. Transversely its extent cannot be made out, owing to the effusion at the left base.

The diagnosis arrived at in this case at the time was ascites, hydrothorax (double), tumour of liver. Malignant? Hydatid? And the prognosis was stated to be unfavourable. He remained in the wards for about nine months, during which time he was tapped on nineteen occasions, about 350 oz. being withdrawn at each tapping. At the end of this time he felt better, and was discharged. He has remained feeble, but still able to move about a little ever since, and during the last fifteen years he has repeatedly come to the Infirmary to let me see him. During all those years he has had absolutely no ascites, and the enlarged superficial veins on the abdominal wall have quite disappeared. His spleen has been getting smaller, so that now it can only be felt deep in the left hypochondriac region when he takes a full inspiration. The liver, however, is yet (March 1897) distinctly enlarged, and can be felt in the right lumbar, right hypochondriac and epigastric regions. It is firm in consistence, its surface is uneven and its lower border round and somewhat nodulated.

Case VIII.—*Supposed Tumour of the Liver—Diabetes Mellitus—Diabetic Cirrhosis*

John B., aged fifty-seven, a gardener, was admitted to Ward XXXI., on November 11th, 1897, complaining of great thirst and extreme weakness, and stating that he had been ill for about three months.

His family history is very good, and he has always lived carefully and soberly. He has been a good deal abroad.

As regards previous illnesses, he seems to have had an attack of cholera many years ago in Canada. He had sunstroke at the Cape, and a few years ago, in Australia, he had a severe attack of influenza. Since then, he says, he has never felt so well. Two years ago he was in this ward for two months, for an attack of pleurisy with effusion. He recovered from this very well.

About six months ago he came to the Infirmary complaining of a severe pain underneath the costal margin in the anterior axillary line. He was also complaining then of great weakness.

On examination at that time a distinct rounded mass about two inches in diameter could be felt at this point.

This mass was evidently connected with the lower surface of the liver, moving up and down with it in respiration, and being distinctly tender on pressure. I believed at the time that I was dealing with a malignant tumour of the liver, but I did not convey my opinion to the patient. I advised him to wear a flannel bandage, and I gave him small doses of mercury and morphia. He did not wish to remain in the ward at that time, and so I asked him to report himself in a few weeks. When he did so I found he was distinctly better, and the tumour was less easily palpated. Feeling distinctly better, he did not come back again to the Infirmary till the date of his admission, when he came complaining of the symptoms of his present illness, viz., diabetes mellitus.

Present illness.—This, he says, began about three months ago, when he noticed a sweetish taste in his mouth, specially on rising in the morning. This was soon associated with thirst and the passage of a large quantity of water. Finding that those symptoms were increasing, and that he was getting weaker and weaker, he came to the Infirmary.

State on admission.—He is about six feet in height, and his weight is 10 stone. His temperature is rather subnormal, and his pulse varies between 60 and 70 per minute.

Alimentary system.—He has great thirst and dryness of the mouth. His appetite and digestion are very good, except for a little flatulence after dinner. His bowels are constipated. On examination, the abdominal parietes over the liver are seen to be slightly prominent. The liver is enlarged, percussion showing that it extends from the fourth interspace above, to $1\frac{1}{2}$ inches below the costal margin. It can easily be palpated, especially in the epigastrium, and its surface feels a little irregular. Its edge feels sharp and a little uneven, and its consistence is evidently increased. The spleen appears slightly enlarged on percussion. There is no ascites, but the superficial abdominal veins are somewhat enlarged.

Urinary system.—Urine, quantity about 60 ozs. Specific gravity varies between 1032 and 1038. It is of a pale straw colour, and has an acid reaction. It contains sugar to the extent of about 2 per cent. There is no albumen, and the urea is about 500 grains daily.

The other systems are practically normal.

Case IX.—*Supposed Tumour of the Liver—followed by appearances of Cicatricial Contraction*

Edward T., aged thirty-six, a lithographer, was admitted to Ward XXXI., January 1st, 1898, complaining of pain in the left

side of the epigastrium, which has been very severe and associated with tenderness, and which is specially marked after taking food. He stated that he had been ill for over a year, but that four months ago his symptoms had got very much worse.

Previous illnesses.—Four years ago he had an attack of erysipelas, during which he said the bowels never moved for ten days. He says that since then his stomach has never been so strong. Three years ago he had an attack very like the present, but it passed off in about a week. As previously stated, he has been troubled with his stomach for over a year, but about a month before his admission he was seized with sudden pain in the epigastric region, rather to the left of the middle line. This kept him off work, and he was treated by fomentations and blisters. A doctor saw him and recognised a tumour in the epigastrium, and for this he was sent into the Infirmary.

On admission he appeared somewhat thinned; his height was about 5 ft. 7 in., and weight 8 stone. Temperature about 99° F., pulse about 90.

Alimentary system.—Tongue somewhat furred. Pain shortly after taking food—sometimes vomiting. Bowels constipated. On examination of the abdomen a distinct area of resistance could be felt in the epigastric region. It was tender on pressure, but, he said, not nearly so tender as it was a month ago. This resistance was found to be the left lobe of the liver, which reached to about 2 in. below the xiphisternum, and which could be distinctly palpated as a firm mass.

The spleen was also slightly enlarged and on both sides "floating kidney" could be made out.

He was treated by rest, small doses of mercury internally and red iodide of mercury inunctions, and he rapidly improved. He was discharged on February 14th and he returned to show himself on February 27th. On that date the left lobe could still be made out about $\frac{3}{4}$ in. below the xiphisternum, and its surface was a little uneven. Otherwise he was feeling well.

In all these cases I was very careful to ascertain that syphilis as a cause could be eliminated. I believe, therefore, that there must have been a localised hepatitis, but whether this has been due to circulatory disturbance, as thrombosis, or to some bile duct or parenchyma inflammation, I cannot say. In the first case (W. L.) the hepatitis has been most extensive and severe, and it has led to the slow cirrhotic change. In the second case it must have, at least, aided the glucose in the blood to set up the process. In the third case there has been some local fibrosis, as shown by the uneven condition of the surface of the liver; whether or not diffuse cirrhosis will follow we cannot yet say.

It would be very interesting in this connection to know in what proportion of cases the healing of liver abscesses, or of liver hydatids, is followed by cirrhotic changes. One would expect that in individuals in whom the nutritive power of the liver has been lowered by alcohol, etc., this sequence would be a common one. This, however, we can best discuss later on.

In this way we can recognise that a cirrhotic change in the liver may be the result of a great variety of causes. It may be the result of irritating substances in the blood, of the portal vein, or of the hepatic artery, as alcohol, syphilis, malaria, chemical products of digestion, glucose, etc. It may be the result of catarrhal or other obstruction in the bile ducts, or of obstruction to the hepatic veins. It may further be the result of the spread into its substance of fibrous tissue, from fibrous adhesions on its surface or from fibrotic cicatrixes in its interior.

But in all those cases we have to recognise one main change, and that is, overgrowth of the connective tissue and relative disappearance of the parenchyma. What we, therefore, have now to do is to understand how this overgrowth of fibrous tissue and relative disappearance of parenchyma can, from the pathological point of view, be explained. To do this, let us consider for a moment the pathological changes which occur in acute yellow atrophy of the liver.

In a typically acute case of this affection, there is a rapid disintegration and disappearance of the parenchyma. The organ becomes, in a few days, diminished to a fraction of its normal size. It is soft and flaccid, so that, laid on a table, it flattens out. Folds are often seen on its surface, it being, as it were, too small for its coat. On microscopic examination, the great change is seen to be disintegration and disappearance of its cells. There is little change in the amount of its interstitial tissue.

Next let us remember that in those instances of acute yellow atrophy in which the course has not been quite so rapid, it has been remarked that whilst the liver cells have as before become disintegrated, and have largely disappeared, a relative increase in the amount of interstitial tissue will be met with. That is to say, what we find is that in the less rapid examples of acute yellow atrophy, the toxic substance, whatever it may be, which rapidly destroys the liver parenchyma, seems at the same time to cause an overgrowth of the interstitial tissue.

Regarding the matter etiologically, morever, we seem to be

corroborated in this view, because we find that toxic substances, which in huge quantity will produce what is practically an acute yellow atrophy, will in smaller continued quantities produce a cirrhosis. As we all know, continued imbibition of alcohol is the commonest cause of cirrhosis. At the same time alcohol, in concentrated form and in huge quantity, has been shown to induce an acute yellow atrophy. Somewhat similar results have been shown to follow the administration of one large, and of continued small doses of phosphorus. Again, in the horse's liver, it is known that the ingestion of a large quantity of lupins causes changes like those of acute yellow atrophy, whilst the continued ingestion of small quantities produces a form of liver cirrhosis.

Now what the toxin which ordinarily produces acute yellow atrophy may be, we do not know, but we are, I think, safe in saying that, like the toxic substances just mentioned, it is one which, in lesser quantities, would produce liver cirrhosis. How, therefore, can we explain such an action? How is it that a toxin is capable, at one and the same time, of causing disintegration and destruction of parenchyma and overgrowth of fibrous tissue?

To understand this, let us consider for a moment in a broad general way the subject of nutrition and reproduction.

Suppose then we take a cell. We know that it lives in a nutrient fluid. We know that it grows and enlarges in this fluid until it reaches a certain size, and then that it reproduces itself by splitting into two, and that these two young cells go on growing and reproducing much like their parent. How does it nourish itself and grow, and how does it reproduce itself? In the first place, we know that the cell is a living structure, and that life means a using up of material. The cell lives by taking in from the fluid around nutrient matter, by building up out of this its own complex tissue, and by using up or oxidising this, and giving it off in the simpler forms of carbonic acid, etc. How it does all this, students of molecular physics have attempted to explain, but we need not put off time with this to-night, because we have other phenomena to consider which are more directly interesting.

Now observe that one small cell not only lives but grows, that is to say, increases in mass. To explain this we must suppose that one cell is capable of taking in and assimilating more than the exigencies of its living renders it necessary to break down or oxidise. Its income is greater than its expenditure, and so it increases in bulk. Now observe that as it increases in bulk its facilities for acquiring nourishment diminish. We know that if

we compare two bodies of similar shape but different size their respective volumes vary as the cube of their diameters, whilst the proportion of their surfaces is as the square of their diameters, that is to say, we find that as the cell increases in size its volume increases more rapidly than its surface. Now of course it is through its surface that it gets its nourishment, as it increases in size, therefore, it is getting less and less nourishment because its surface is becoming less and less in proportion to its mass. Finally, a time comes when it cannot get a sufficient supply. What happens then? It simply breaks down into two or more pieces. But these smaller pieces have each a large surface in proportion to their mass. Hence they at once begin to grow, and form cells like their parent. Thus, then, when the nutrition of the original cell has failed, reproduction takes place, and the death of the individual means the life of the race.

But now notice that the period of growth or life of the individual cell must vary (1) with the metabolic activity of the cell, and (2) with the amount of nutrition around. With the same amount of nutrition around, the greater the metabolic activity (wear and tear) the shorter will be the period of growth and of life, *i.e.*, the earlier will reproduction tend to occur. Also, the metabolic activity remaining the same, the less the amount of nutrition around, the shorter will be the period of growth and of life of the cell, the earlier will reproduction occur. In this way, then, excessive metabolic activity and lessened amount of nutriment each tend to lessen the period of growth or life, and correspondingly to favour reproduction.

Next observe the inter-relationship between growth and development. Growth means increase in bulk, development means change in structure. We have the growth of cartilage, and we have the development of cartilage into bone. Hence as development occurs on the cessation of growth, development has been regarded as antagonistic to growth.

Development, however, is not antagonistic to growth as reproduction is; it is better described as a substitution for it. In a young growing organism the trophic power is used in increasing bulk, *i.e.* for growth, with the completion of growth this trophic power is made use of to cause change in structure, *i.e.* for development.

Hence it is evident that reproduction is antagonistic both to growth and development. But the important point for us to

note is, what must happen in the case of a developed organism or cell, where failure of nutrition, relative or absolute, has occurred. It is evident that reproduction must occur, but the impairment of nutrition continuing, it is evident that this reproduction will be the reproduction of organisms or cells less complex and less developed. Development and complexity will be sacrificed to numbers, quality will be sacrificed to quantity, for the continuance of the life of the cell or organism is the last thing which nature yields.

And now follows the important deduction, viz., that as the result of absolute or relative failure of nutrition, the more complex or developed cell may suffer more than the less complex or less developed one.

To explain this, let us suppose that we have in the same nutrient fluid two cells, A and B. Let us suppose that they are endowed with an equal amount of nutritive energy, but let us suppose that whilst in A's case, the metabolic energy is expended rapidly in causing it to grow and develop into a complex developed and actively working cell organism, in B's case the energy is expended much more slowly, because it has to grow into a comparatively simple inactive cell organism.

In A's case, growth, complex development, and reproduction, let us suppose, will be completed in a week. In B's case, growth, simple development and reproduction will be completed in a month.

Let us next suppose that, as the result of some irritant, some toxic substance, the metabolism or wear and tear of A and B is stimulated into increased activity, the amount of nutritive material around remaining the same. It is evident that the fate of A will be the harder, for the increased demand for nutrition and oxygen will be more acutely felt. Reproduction, at the expense of development and growth, will, time after time, more and more rapidly occur, and therefore less and less complex cell products will be produced, so that the form of the original complex cell will soon be lost. With B, on the other hand, this increased demand for nutrition and oxygen will have a much less deteriorating effect. Reproduction will of course go on much more rapidly, but as the parent cell was comparatively simple, the new formed cells will be little less so, and so the form of the original cell will be in great measure preserved.

Given, therefore, a certain amount of toxic irritation, we can understand that whilst in the case of the more elaborated and

complex liver cell, the results may be rapid degeneration and even death, in the case of the less complex connective tissue cell the results may be the production in great numbers of cell-fibres little less complex.

In the more rapid examples of acute yellow atrophy, therefore, I would suggest that the toxic substance is tremendously powerful. It therefore disintegrates and destroys the liver cells, and before distinct overgrowth of the fibrous tissue has had time to occur, the patient has died.

In less rapid examples, on the other hand, I would suggest that before the disintegration of liver cells has produced a fatal termination, the interstitial tissue has had time to increase.

Lastly, in cirrhosis of the liver, the toxic substance has acted so mildly that with the destruction and disappearance of liver cells the interstitial tissue has increased to a marked degree. In all those morbid conditions a further important pathological change is apt to occur, viz., proliferation and new formation of bile ducts. This requires explanation, but its consideration can better be understood after the subject of cancer and cirrhosis has been discussed.

Case X.—*Alcoholism—Pains over liver—Ascites—Death from coma—Cancerous cirrhosis*

C. M., aged fifty-three, at present a clerk, but for many years a traveller in the wine and spirit trade, was admitted to Ward XXXI., December 28th, 1897. His complaints were pain in, and swelling of, the abdomen, swelling of the legs, and great weakness. The pain he had noticed for the last twelve months, but it only got unbearable about a month ago, when the swelling of the abdomen appeared. The swelling of the legs began five days ago.

His family history is very good, and his surroundings at home quite satisfactory, but for many years, while engaged in the spirit trade, he had been constantly taking drink. He had had a slight attack of pleurisy six years ago, and influenza eighteen months ago. Previous to this influenza attack, he had considered himself perfectly sound in every way.

State on admission.—Height, 5 ft. 8 inches; weight, up till a year ago, 12 stone 10 lbs.; at present he weighs 10 stone 7 lbs., but much of this is undoubtedly due to ascites. His muscles are small, and his abdomen is greatly distended, his legs also are swollen. No jaundice. Pulse 72, temperature normal.

Alimentary system.—Tongue slightly furred, appetite fairly good, but pain after taking food. Bowels fairly regular.

Abdomen much enlarged from ascites, circumference at umbilicus 38 inches. The liver dulness begins above at the fourth rib in the nipple line. In the epigastrium and right lumbar region its surface can be felt hard and slightly rough; its edge is somewhat rounded and uneven. In the nipple line its lower border can be felt about 1 inch below the costal margin, in the middle line its lower border is about $3\frac{1}{4}$ inches below the ensiform cartilage, and its left lobe can be felt disappearing under the costal margin opposite the junction of the eighth with the other costal cartilages.

The spleen is enlarged. It can be distinctly felt on palpation under the left costal margin. On percussion its area of dulness is $7\frac{1}{2} + 5\frac{1}{2}$ inches.

With the exception that his urine deposits brick-red urates, with usually a copious covering of the vermilion coloured purpurine, all his other systems are fairly normal.

In the ward this patient gradually got worse in spite of treatment. He obtained temporary relief as the result of paracentesis on two occasions. Although for the last three weeks of his life the ascites did not increase, he got rapidly weaker. On the 31st of January his temperature, which had been subnormal, rose to 100° F., his breathing became more rapid, and showed the Cheyne-Stokes character, he became distinctly comatose, and he died at 11.30 P.M.

Post-mortem report.—Body emaciated, no jaundice. Peritoneum contained about 100 ozs. of slightly turbid fluid. Liver weighed 4 lbs. 12 ozs.; its surface and section showed the appearances of common cirrhosis. In addition, however, especially in connection with the left lobe, it showed diffused malignant growths. Portal vein blocked by a large firm thrombus, bile ducts normal, gall-bladder full of dark coloured bile. Spleen enlarged. No evidence of malignant disease elsewhere.

A careful microscopic examination of the liver and other parts in this case was made by my clinical clerk, Mr James Miller, and the following is his report:—

Tissues taken—

I. Liver.

1. Part showing only malignant disease.
2. Part showing only cirrhosis.
3. Part showing both.

II. Lymphatic glands from behind the liver.

These tissues were fixed in saturated $HgCl_2$, cut in paraffin and stained, some with HæmR and O, others with Maun's methyl blue and eosin.

1. Part showing only malignant disease.

(*a*) Nodules of cancerous tissue varying in size, in shape distinctly rounded.

(*b*) Fibrous tissue in thick bands between the nodules. This fibrous tissue is as a rule well formed, but in places, largely cellular.

(*c*) Normal liver tissue in small amount at the periphery of the cancer nodules, evidently pushed to one side, and undergoing atrophy. In other places normal liver tissue is seen in small islands, surrounded by a zone of small bile ducts radiating from them.

In many places the cancer nodules are undergoing degeneration, becoming broken up into a granular debris. The cancer cells are large, with large nuclei. They are not arranged in columns, like normal liver cells, but in irregularly rounded masses of a few cells, with fibrous tissue between. In many, especially in the smaller solitary nodules, the cells seem to be arranged as in a gland acinus, with a central lumen. The main difference between cancer and liver cells lies in the fact that the cancer cells contain highly refractile masses, staining bright red with rubin, and a bright crimson with eosin.

2. Part showing only cirrhosis.

The cirrhosis is as a rule fine, tending to be monolobular, but in places it is coarser. The sections show the usual appearance of cirrhosis. The fibrous tissue is as a rule cellular, in places very cellular. It is also very vascular. There are considerable numbers of small bile ducts in the fibrous tissue.

3. Part showing both (see plates).

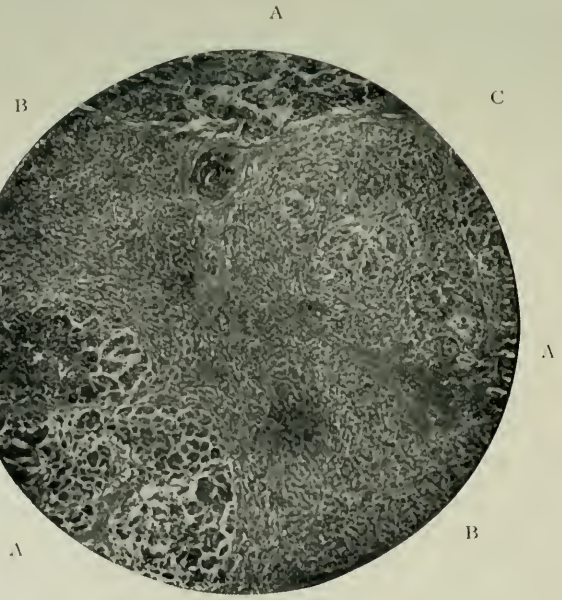
Rounded nodules of cancerous cells appearing—

(*a*) In the midst of a normal liver lobule. It pushes aside the normal liver tissue, and causes flattening and atrophy of the cells.

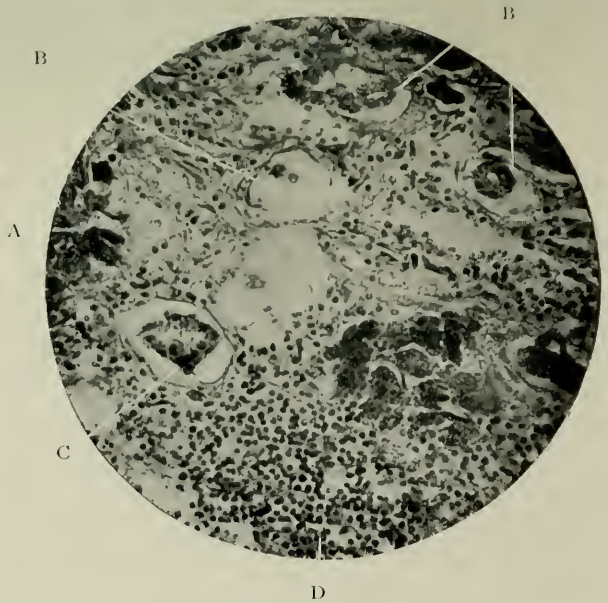
(*b*) In the fibrous tissue also as rounded nodules, varying in size from a few cells to as large as a liver lobule, or larger. They show also the gland-like arrangement. In places the cancer cells are degenerating.

The normal liver tissue is seen pushed aside by the cancer nodules in many places. Islands are also seen, as previously mentioned, surrounded by radiating bile ducts of small size, just as if the normal liver tissue were melting away into bile ducts. In these islands of liver cells distinct transition can be traced with a high power from the normal liver columns to the small bile ducts. The columns of liver cells seem to divide, to open up, and gradually losing their characteristic staining reaction, to become true bile duct cells, ceasing to stain with the rubin, and staining merely with the hæmatoxylin. The liver cells seem to lose the golden yellow pigment in the process, this becoming discharged into the surrounding fibrous tissue. In places this pigment can be seen aggregated into clumps, indicating the position of an island of liver tissue which has melted away.

PLATE VIII.—TO ILLUSTRATE DR JAMES' PAPER.



A=CANCER NODULES.
B=NORMAL LIVER TISSUE.
C=FIBROUS TISSUE CONTAINING SMALL CANCER NODULES AND BILE DUCTS × 26 DIAM.



A=CANCER NODULES.
B=BILE DUCTS.
C=CANCEROUS BILE DUCT.
D=CELLULAR FIBROUS TISSUE × 250 DIAM.

The fibrous tissue is much more cellular in the cirrhotic part, and in the parts in process of transition. It is much more fibrous in the purely malignant portions. It is probably more vascular in the cirrhotic portions, although hæmorrhages are not uncommon in the degenerating cancerous areas.

The new bile ducts are distinctly more numerous in the areas of transition, especially round the islands of normal liver cells, previously mentioned. Many have the structure of a normal bile duct, *i.e.*, cubical epithelium with central nucleus, staining with methyl blue alone. But many show small masses in their protoplasm, staining with eosin, exactly like the masses seen in the cancer cells. In fact, it is sometimes difficult to distinguish between the small bile ducts and small cancer nodules in the fibrous tissue, the only difference being the evident lumen in the case of the bile ducts.

II. The lymphatic glands from behind the liver show no trace of malignant disease.

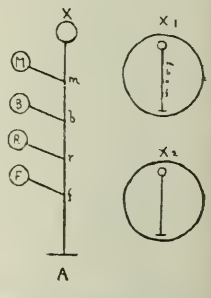
To trace the connection between liver cirrhosis and liver cancer, let me now briefly discuss the etiology of tumours. A new formation has been defined as follows :—"The excessive production of some tissue naturally existing in the living body during fœtal or adult life, either locally, where such tissue is normally present, or in some part of which it is not an actual constituent." But now the question is—How can a new growth presenting embryonic structure and characters be started among the adult tissues ? The best known hypothesis on this subject is Cohnheim's.

In Ziegler's *Pathological Anatomy*, p. 250, it is stated : "We are not to refer the actual development of the tumour itself to the embryonic period, but are to attribute its appearance in later life to the persistence of germinal embryonic tissues in the otherwise mature organism. A tumour takes its rise in what we might call a belated rudiment, a focus of formative embryonic tissue which has not been utilised in elaborating the normal tissue of the part, and so has lingered on unchanged." Cohnheim therefore defines a tumour as an atypical new formation starting in a latent embryonic rudiment. The tumour germs, consisting as they do, of embryonic cells, may be very small, and so escape observation. It is even conceivable, he thinks, that the germinal cells may be quite unrecognisable among the ordinary physiological elements of the part. They may linger on for a long time inactive. It is only when they are favoured by the external conditions such as the supply of nutriment, and their relation to

the surrounding tissues, that they begin to multiply and to form a tumour.

This is Cohnheim's view, but although the persistence of embryonic structures in teratological formations may seem a point in favour of it, I have no hesitation in saying that it is unsatisfactory and in a way unphilosophical. Let me illustrate this by an analogy.

As we all know, in the sociological organism the criminal is mainly the man who, from the ethical point of view, is not up to the standard of the age in which he exists. He is one who, in his endeavours to obtain subsistence, makes use of means which, legitimate enough in a bygone barbarous age, are not up to the ethical standard of the age in which he lives. Now, according to Cohnheim's theory, a criminal would be a man, who, born some centuries ago, has lived in a sort of hibernating condition till the present time, and has now burst out into full activity. The unphilosophical character of this theory is at once manifest. We know that every living being is a potential criminal, and that whether or not he or she becomes an actual one, depends on constitution and surroundings. We hold, therefore, that any tissue may develop a tumour—whether it does so or not depends similarly on constitution and surroundings. Here let me quote the theory which I put forward in this Society sixteen years ago. "Is it not more probable that, instead of there being in the adult a piece of embryonic tissue latent, there is taking place a continual transition process of simple cells into the various differentiated tissues, similar to what takes place in the embryo, only much more rapidly. In this transition process, there will be stages in which the tissues formed exactly resemble the embryonic ones. Suppose that this is the case, and suppose that at one or other of these stages reproduction occurs at the expense of nutrition, we should have a mass of tissue, more or less resembling the embryonic, *i.e.*, a tumour, produced.



Let me illustrate what I mean by a scheme. Let the line A X represent the evolution progress from the primitive protoplasm to man. In this progress the stages of fishes (*f*), reptiles (*r*), birds (*b*), and mammals (*m*) have been passed through, and from each of these have sprung fishes (*F*), reptiles (*R*), birds (*B*), and mammals (*M*) as we now see them

around us. But now let us take man as represented by the large circle X1. Each new human being passes in nine months or thereabouts from the protoplasm stage (ovum) into man. In doing this it has to pass through stages, fishes *f*, reptiles *r*, birds *b*, and mammals *m*, in exactly the same manner as in A X, but, of course, immensely more rapidly.

Still further, let us take a tissue in man, *e.g.*, a liver cell, and let us call it X2. Each liver cell lives a certain time and has, before disappearing, to reproduce itself. Now we may feel sure that each young liver cell, in growing and developing, passes in its turn through stages similar to those which it has passed through in embryonic and fœtal life X1, but proportionately again much more rapidly.

Remembering how that when absolute or relative failure of nutrition occurs, reproduction will ensue at the sacrifice of growth and development, and that thus less and less elaborated and developed structures must in time be produced, we have to look on any factor which entails premature wearing-out of the trophic power of the liver cells as a cause of these cells retrograding and in time taking on embryonic characters, that is, becoming cancer cells. In our patient this factor has been alcohol. This consideration enables me to emphasise two very important points.

I. That cancer tissue may not be absolutely like embryonic tissue. This can be best shown by a reference to A X, for here something analogous can be recognised in the fact that fishes *F*, reptiles *R*, birds *B*, and mammals *M*, are not exactly like what they were at *f*, *b*, *r*, or *m*, *i.e.*, at the stages through which man has passed in evolutions. Fishes, reptiles, birds, etc., as we now see them, are all direct developments in certain directions from these evolution stages, but are not identical with them. So also may cancer cells be regarded as developments from embryonic cells at different stages.

II. That to explain the beginnings of growth of cancer cells amongst other hepatic cells as the result of absolute or relative impairment of nutrition, we have to postulate the existence of something which leads to a disturbance of the balance of nutrition among these cells. Thus we do not find cancer developing in the liver in every case in which some cause leading to premature wearing-out of its nutritive power is existent. Nor do we find it apparently in cases where an impaired nutritive condition exists as the result of obstructed portal veins, although in such cases

increased number of bile ducts and relative increase in the amount of fibrous tissue are to be found. Something there must be, therefore, to disturb the balance, and this something may be of endless variety. Just as the manifestation of syphilis may be in one individual an aneurism, in another locomotor ataxy, in this one a waxy liver, in that one a myelitis, etc. etc., all depending on different determining factors, age, constitution, injury, strain, exposure, peripheral irritations, etc., so will it be with the onset of cancer in this or that part of the liver in individuals in whom what we call impaired trophic power exists.

To revert to our evolution diagram A X, something analogous can be recognised in the factors which, acting at *f*, *r*, *b*, and *m*, have caused certain individual organisms to branch off to *F*, *R*, *B*, and *M*, *i.e.*, to fishes, reptiles, birds, and mammals, as we now see them, and others to go on growing and developing into man.

Next, let us consider what embryologists tell us as regards the development of the liver. To do this I quote liberally from Hertwig's Text-Book :—

"From the ventral wall of the duodenum a little sac grows out, and this shortly afterwards becomes doubled. This beginning of the liver is metamorphosed rapidly into a tubular gland with numerous branches. These tubes early become joined together to form a fine network, since the primitive hepatic tubes send out numerous lateral buds. Those in mammalia and in man are solid. Embedded in the embryonic connective tissue of the ventral mesogastrium, they grow out into solid cylinders. Inasmuch as they grow towards one another, and where they meet, fuse, there arises a close network of solid hepatic cylinders in the common connective tissue matrix. Simultaneously with the formation of this epithelial hepatic network, there is formed in its meshes a network of blood-vessels, from the vena omphalomesenterica. This vessel gives off numerous shoots, which in turn give off lateral branches, which unite with one another in a manner corresponding to that of the hepatic cylinders. Eventually the network of hepatic cylinders is metamorphosed in two ways—one part becomes the bile duct and its branches ; the other furnishes the secretory parenchyma of the liver cells." That is to say, in the development of the liver we recognise three main stages—(1) hypoblastic cells, (2) bile duct, (3) parenchyma.

The general conclusions which we can reach from these considerations, therefore, are :—

1. That when as the result of some toxic substance, or of some other injurious condition, there is a tendency to reproduction at the expense of growth and development, and consequently a tendency to retrograde processes in the liver tissue, the formation of new bile ducts will be a usual accompaniment. The new bile ducts simply represent a certain stage in the retrograde change. Let, however, this retrograde process be pushed further, then the cells will become as regards simplicity of structure and function still more embryonic. We shall therefore have a cancer.

And here comes a very interesting point. Looking at the matter from this aspect, we should expect that in the transition backwards from hepatic cells to cancer cells we should be able to recognise a transition through the cells of the new-formed bile ducts. Now an examination of sections from the liver affected with cancerous cirrhosis (page 15) reveals, I think, that this hypothetical change has been actually taking place. As Mr Miller has pointed out in his report, it is seen that whilst at one part the liver cells are, as it were, melting down into new bile ducts, at another part the cubical new bile duct epithelium is taking on the characters of cancer cells.

In connection with those cases of cancer cirrhosis, the question has often been discussed as to whether the cirrhosis or the cancer occurs first. In our case it is, I think, very evident that it has been the cirrhosis. In the purely malignant portions the fibrous tissue is distinctly more fibrous, *i.e.*, it is the older.

Let me now make a *résumé* of the conclusions which I would wish drawn.

I. In the very rapid cases of acute yellow atrophy, the toxin is so tremendously powerful that it destroys the liver cells before reproduction, and consequent increase of the fibrous tissue occurs to any appreciable extent. In the less rapid cases, the toxin acts rather less intensely, so that before the disintegration of the liver cells is sufficient to kill the patient, the interstitial tissue has time to reproduce itself to some extent. In both cases new-formed bile ducts are an associate.

II. In cirrhosis of the liver, the toxin acts more mildly. Time is therefore allowed for more marked retrograde changes to occur. The interstitial tissue has multiplied to an extreme degree, and the liver cells are functionally and structurally

degenerated. New-formed bile ducts are to be looked for, and, bearing in mind the factors which lead respectively to the biliary and portal forms of cirrhosis, we can presume that they should be more marked in the former than in the latter.

III. In cirrhotic cancer of the liver, the toxin acts also more mildly. It gives time therefore for the retrograde changes described above to occur, viz., fibrosis, degeneration and disappearance of liver cells, and formation of new bile ducts. But the retrogression has gone further, for the epithelium lining those bile ducts has become still more embryonic in character. The cells, indeed, have practically become cancer cells, and are nourishing and reproducing themselves as such.

In this paper I have limited myself mainly to the pathological processes concerned in cirrhosis and cancerous cirrhosis of the liver. It will be evident, however, that regarding these processes as being essentially due to disturbances from the normal in the fundamental processes of growth, development, and reproduction, what I have said applies with little modification to fibrotic processes and to new growth formations in general. It is interesting to note that some such transitions as I have referred to, have been believed to exist by other observers. Thus, in cirrhosis of the liver, Hamilton describes the transformation of liver cells into spindle cells, and then into fibrous tissue. Although I have accounted for the fibrosis in another way, yet the theory which I have put forward does not altogether negative this as another possible event. Anfrecht[1] speaks of a similar transformation of liver cells into fibrous tissue, and refers to the new-formed bile ducts in acute yellow atrophy, etc., as a "rückbildung." Also Thorel,[2] in connection with a case of cancerous cirrhosis, attributes in an interesting and similar way, the cirrhosis and the cancer to the same irritant. What I desire, however, to emphasise in this paper, is the unity of all those pathological processes, inasmuch as they all can be interpreted as being essentially the result of disturbances in the normal tissue processes of growth, development, and reproduction.

In conclusion, I have to express my sense of indebtedness to Dr Robert Muir and to Mr James Miller for their kind assistance in connection with the pathological subjects referred to.

[1] *Real-Encyclopädie der gesammten Heilkunde.* Band xiii.

[2] *Die Cirrhosis hepatis Carcinomatosa, Beiträge zur patholog. Anat.* (Ziegler). Band xviii., 1895, page 498.

Meeting VII.—May 4, 1898

Dr James Carmichael, *Vice-President, in the Chair*

I. Exhibition of Patient

Dr Scot Skirving exhibited—

A CASE OF ECTOPIC PERINEAL TESTICLE.

1. Patient after operation.
2. Specimen.
3. Cast of parts before operation.

Patient, a boy, J. W., age 5½ years. Brought to New Town Dispensary suffering from painful micturition, particularly on first getting up in morning.

History of kick on perineum from another boy three weeks previously. Above symptoms did not appear till a fortnight after this accident, when father's attention drawn for first time to a swelling in perineum.

On Examination.—Oval swelling on right side of middle line, about size of walnut, extending from base of scrotum nearly to anus—slightly tender. R. testicle found absent from scrotum. Other testicle normal.

Operation.—Incision made over swelling. Interesting condition found, viz., tunica vaginalis found which was continuous with general peritoneal cavity, *i.e.*, the condition seen in an ordinary congenital hernia minus the presence of gut or omentum.

This pouch of peritoneum had, therefore, come down, going past the scrotum, but not entering it at all. The testicle was normal but there was considerable thickening of the tunica where it was attached to the testicle.

It was found impossible to transplant testicle into scrotum in this case, there not being room enough in right half of scrotum. It was therefore removed, the incision extended and Macewen's Radical Cure performed. Patient showed a sound cicatrix and an entire absence of symptoms.

II. Exhibition of Instrument

Dr William Taylor exhibited a new instrument for the treatment of neuralgia, called the Electro-thermogen, of which the following is Dr Taylor's description :—

I was sitting at a window on a summer afternoon watching the players at a game of tennis. The window was of plate-glass, and the surroundings comfortable; but I was conscious of a very slight draught from the corner where I sat. This was rather refreshing than otherwise, and that, together with the interest of the game, made me disregard it. In the evening I was suddenly seized with a violent attack of neuralgia affecting the side of my face which was nearest the corner. It was a new experience. The pain was superficial, and I tried to relieve it by superficial remedies, all of which failed, and I wondered that a cause so slight could be capable of producing agony so intense. By and bye my eye became affected, whether from the disease or the remedies I could not tell, but I thought I should become blind as well as mad. Under the influence of these mingled feelings I spent half the night; but at length fell asleep. In the morning I awoke in a profuse perspiration and absolutely free from pain, so that I spent the day forgetful of it. In the evening my enemy returned reinforced by a contingent ten times worse than himself, and this he continued to do on successive evenings with diabolical punctuality, making my days apprehensive and my nights hideous. During these attacks, which I yet shudder to think of, internal remedies were powerless, and the only alleviation I obtained was from heat. Especially *dry* heat. Steam relieved for a time; but the resultant moisture produced a feeling of cold, which, although slight, was sufficient to renew the pain, and hot dry flannel had to be substituted, which, with often repeated applications, gave relief.

This experience presented to me a definite problem: "If a current of cold, and more or less moist air be capable of setting up neuralgia, why should not a current of dry hot air cure it?" and I set myself the task of producing a hot dry air current. The proposition was simple, its solution difficult. I found that air is not easily heated and very rapidly cooled, so that it cannot be carried far from the source of its heat. My first device consisted of a piece of brass tubing rolled into a flattened coil and placed upon a blow-pipe flame. Through this, by means of bellows, a current of air was forced, which became heated in transit and emerged hot at the free end. The tubing was difficult to coil, and the process was tedious and expensive. It

was superseded by a casting which I had made from a design suggested by the plan of the "walls of Troy" (fig. 1)—circular in shape, and composed of a tortuous air channel. It is efficient but bulky, and very difficult to warm up, and when hot difficult to handle. I employed it with success in an obstinate case of torticollis, but the patient had to come to *it* as *it* cannot conveniently be taken to the patient. It is not suitable for the treatment of limited areas, such as have to be dealt with in facial neuralgias, and the working of the bellows is very tiresome.

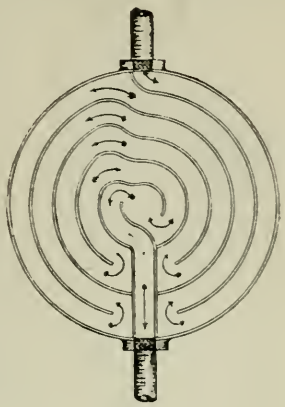


FIG. 1.

Description of the Electro-Thermogen

The electro-thermogen consists of two parts (fig. 2)—one of metal, the other of glass. The metallic portion consists of a shell containing a rotatory fan (fig. 3), the clock-work which actuates it, and a small air chamber above the fan. The glass portion consists of two tubes, one within the other. The one end of the *outer* tube is inserted into an opening on the top of the air chamber, the other end is free. It

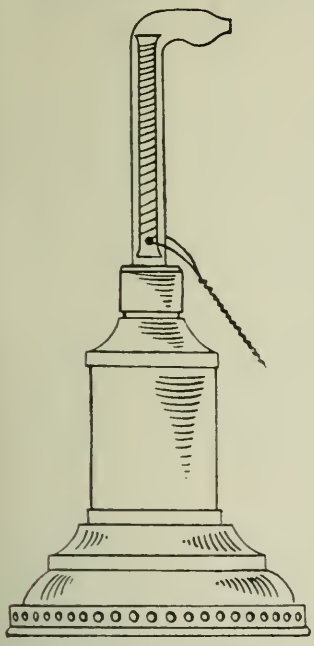


FIG. 2.

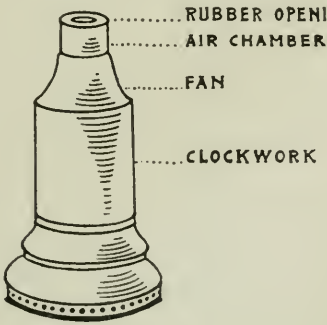


FIG. 3.

may be bent or straight, and may or may not terminate in a

contracted nozzle. *Different forms.*—Close to the free end there may be an expanded portion for the reception of substances suitable for inhalation or as auxiliaries in the treatment of localised pain. These may be inserted by means of a suitable pipette. The *inner* tube is wound round with a spiral of fine platinum wire, the ends of which are connected with terminals fixed in the outer tube. To these are attached insulated wires which convey a current of electricity through the platinum wire, of a strength sufficient to make it hot. When the tubes are placed in position and the clockwork wound up, the fan revolves at a rapid rate, and sends through the heated tubes a current of air which in transit becomes hot and emerges at the mouth of the outer tube in a condition suitable for applying directly to the affected part.

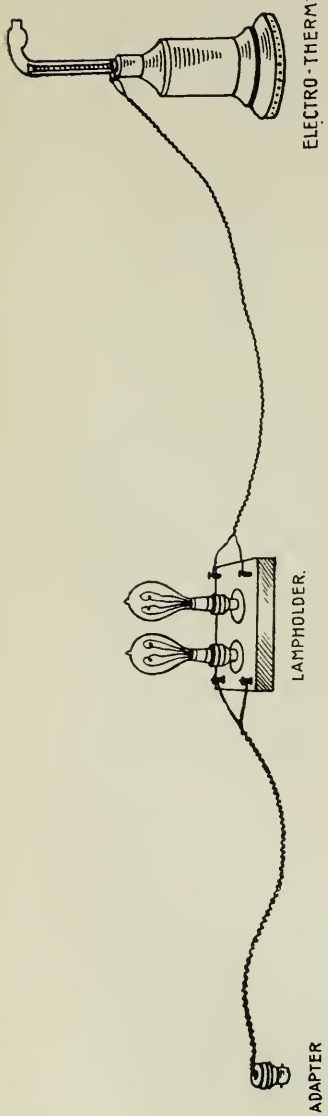


FIG. 4.

Intermediate between the source of electricity and the electro-thermogen is placed a lamp-holder, supplied with lamps of such calibre as to interpose a suitable resistance (fig. 4). By means of these the temperature can be regulated.

I do not know to what degree of heat the temperature can be raised. I tried it with my own thermometer, and found that it went above 212° and burst the bulb. This satisfied me as to its capabilities.

In cold climates persons can endure a far lower temperature if the air be still than if it be in motion, so that the effect experienced is not regulated so much by the degree of cold as by the successive accession of cold particles on the surface, as when a gentle breeze is blowing, hence the detrimental effects

of slight draughts. With heat a similar rule holds good. In a still temperature of 260° a beef-steak can be cooked in thirty-three minutes, but if air of the same temperature be blown upon the meat by bellows, it can be cooked in thirteen minutes.

In accordance with these facts, I have found that in treating neuralgia a moderate current long continued is far more effective than a more intense current of shorter duration. The almost invariable remark of patients when so treated is, "That is very soothing," and no more welcome remark could be made, for anything that is to benefit one suffering from neuralgia must be of a soothing nature.

In connection with this matter, I have had what for me was a somewhat exceptional experience in discovering the existence of thermal anæsthesia in neuralgic areas, so that the patients do not feel either the soothing or the burning effect of the hot air current. In one case, that of a lady aged 22, who had suffered from neuralgia for thirteen months, the affected spot was one inch distant from and in line with the left eyebrow, a minor degree of pain extending along the infra-orbital space. On applying the hot air current, I was surprised to find that she made no response. I increased the heat, but still she did not seem to feel it; but on applying it to parts in the immediate vicinity, she at once winced. After a time the surface of the affected spot became blanched in colour, and I told the patient, on taking her leave of me, that if the part blistered, she was to return and let me see it. This was at 10 A.M. She returned at 2 P.M. with the cleanest, most perfect blister I ever saw. It was the size and shape of a half-walnut, and filled with serum of a deep orange colour. There were a few smaller blisters on the upper part of the cheek beneath the eyelid. On questioning her as to the pain, she replied, "I felt no pain, nor do I feel any now." The neuralgia has not returned, although six weeks have elapsed.

Another patient, aged 20, had suffered for eleven months from neuralgia affecting the left ear. The auricle was tumid and so painful that she had been unable for months to rest that side upon a pillow. Thermal anæsthesia was present, and the result of the application of hot air was a crop of small blisters clustered round the meatus. This was after five

applications; but before the blisters appeared the pain had gone, and she was able to sleep on the affected side. There was deafness on that side. The first day she could only hear the ticking of my watch when it was held close to the ear. At the end of the fifth day she could hear it three inches away.

This has led me to hope that in the treatment of cases of nerve deafness where there is no actual lesion, the electro-thermogen may be of service. Minor degrees of thermal anæsthesia have been present in most of the neuralgias I have treated, and I may say that every application of the hot air which I have made for the relief of pain has been successful.

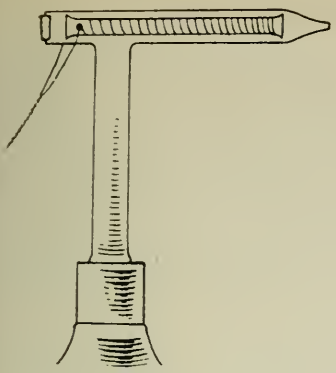
The unexpected sometimes happens. A medical friend had suffered from toothache for three days, and he informs me that five minutes application of the hot air current by means of the electro-thermogen cured him. This was before the commencement of the winter session. When I last saw him, at the end of March, the toothache had not returned. Such a result I could never have anticipated.

Another medical friend informs me that he has found it of benefit to a patient affected with twitching of the facial muscles. This was also an unexpected relief.

One evening a lady entered my study complaining that the act of swallowing caused her great pain on the right side of her neck. She informed me that she had suffered from a relaxed throat, which was better, but she appeared to be considerably run down. I asked her to swallow a mouthful of water, and in doing so the muscles of her lean neck immediately jerked out in painful and continued spasm. I applied the hot air current for ten minutes and she swallowed more easily. Again for ten minutes, a still further improvement, then again, and at the end of half an hour she could swallow without wincing, and she went away happy. The treatment was repeated on the two following days for minor attacks, but no return of the spasm occurred after the third day.

I have not tried it in any case of uterine disease, but judging from the good effects of steam locally applied in these affections, they seem to offer a promising field.

In diseases of the respiratory organs the electro-thermogen



FOR CAUTERIZATION

FIG. 5.

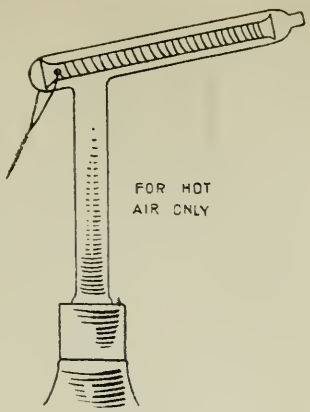
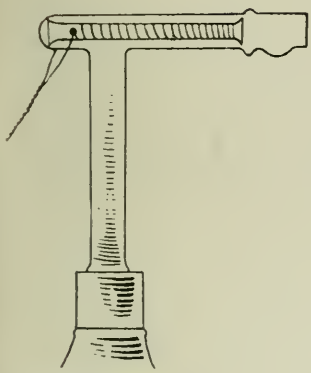


FIG. 6.



FOR INHALATIONS

FIG. 7.

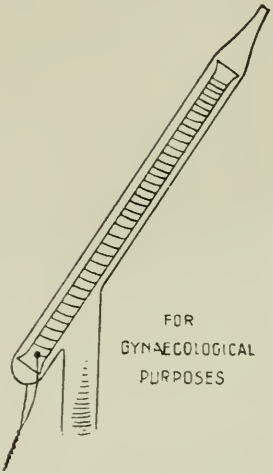
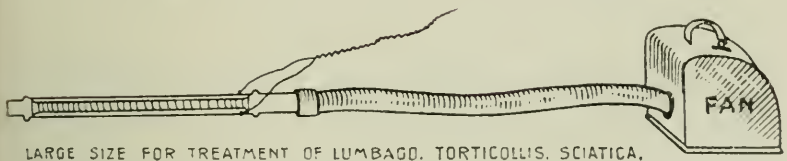
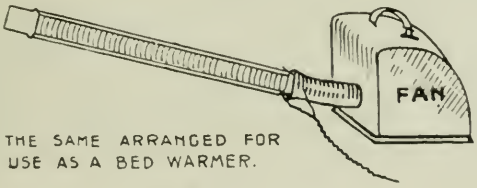


FIG. 8.



LARGE SIZE FOR TREATMENT OF LUMBAGO. TORTICOLLIS. SCIATICA,
AND AFFECTIONS OF LARGER JOINTS.

FIG. 9.



THE SAME ARRANGED FOR
USE AS A BED WARMER.

FIG. 10.

has been of use. It has given temporary relief in a case of gouty asthma and in one of clerical sore throat, but my experience of such cases has been too limited and of too short duration to warrant me in speaking of permanent results. The chief advantages of this treatment appear to be that the substances administered are volatilised and conveyed to the air passages by dry heat without the accompaniment of steam, which is less penetrating and always leaves a disagreeable moisture about the face and neck.

By the kindness of Dr Allan Jamieson I have been placed in possession of an unexpected addition to the list of ailments for which the electro-thermogen may be used. He has drawn my attention to an article in *La presse Medical* for Oct. 30, 1897, in which Mr Hollander of Berlin relates how he treated a case of lupus of the face by air heated to the temp. of 300°. For this purpose he employed a gas flame, in which was placed a pointed tube which became very hot. The air was driven through this by means of an indiarubber hand-bellows, and so heated. It produced an escharotic effect with great exactitude, and without metallic contact, and without encroaching as steam does on the healthy textures adjoining (figs. 5-10).

III. Original Communications

1. ACUTE PNEUMONIA OF CHILDHOOD

By James Carmichael, M.D., F.R.C.P.Ed., University Clinical Lecturer on Disease in Children; Physician, Royal Edinburgh Hospital for Sick Children

Acute pneumonia is a disease which bulks largely in the mortality of children in this and other countries. The disease, not only on account of its frequency, but from the varied and complex nature of its clinical features, is one of great interest. In recent years much has been done to elucidate the subject both from a clinical and pathological standpoint. Bacteriology has contributed largely to our knowledge of the disease. Its etiology, whether in the adult or the child, is essentially the same, and yet how dissimilar are the clinical pictures at the different periods of life. I purpose to give a short outline of our present knowledge of the disease as met with in

early life, based mainly on hospital experience during late years.

Looking back to the earlier writings on the subject and the observations of such men as Valleix, Billaud, Trousseaux and Bouchut, we find wonderfully accurate accounts of the symptomatology of the disease, and also a description of the naked-eye appearances of the lungs on section in its various forms. From want of pathological knowledge based on microscopic investigation, these older authors vainly tried to classify the pneumonias in different ways. Legendre, Barthey, and Bailly divided them into two varieties, based upon the possibility of inflating the lungs and applying the hydrostatic test. The more modern division of the disease into lobar and lobular varieties still holds good as a general and rough classification, but is only serviceable as giving us an idea of the extent of the disease and the general physical condition of the lungs, without affording any information as to the real pathological condition. In the present day it is wonderful how microscopic investigation and bacteriological research have cleared up and simplified our knowledge of the subject. Quite a flood of light has in recent years been thrown in upon the disease, and its infective nature pretty clearly established, so that a new classification of the various morbid conditions met with according to our present knowledge would seem to be called for. The rôle which micro-organisms play in the disease, if not the main factor, is certainly a very important one, as without doubt the toxæmia resulting from their growth and development would appear to be not only the main factor in the production of the constitutional symptoms, but often the principal cause of danger to life. It is a well-known fact that pneumonia in the constitutional symptoms are often out of all proportion to the severity or extent of the local lesion in the lung. The frequency of cerebral symptoms which often occur when only a limited portion of lung is affected, such as the apex, are often associated with profound toxæmia. Other effects of the toxin on the nervous system often manifest themselves particularly on heart centres, where rapid death may take place from heart failure, due apparently to this cause rather than any direct action of the poison on the heart muscle, although the effect of a high temperature, before the crisis, no doubt directly tends to weaken the heart itself. Another cause which appears to produce heart failure has been lately pointed

out by Bollinger,[1] who has noticed the occurrence of oligæmia, due in part to the drain from the blood resulting from the exudate, and in this connection he notes that leucocytosis is a favourable prognostic as indicating new blood formation. Another point of interest with reference to the rôle played by micro-organisms in the disease is in regard to its type and duration. In pneumococcus pneumonia the disease is of short duration and the crisis generally well marked. Whether this be due to the fact that the life-history of this organism is a comparatively short one, or that an anti-toxin is more rapidly produced than in the case of many other micro-organisms associated with other forms, we do not as yet know.

Classification.—The most rational and scientific mode would seem to be that founded on the pathological anatomy and bacteriology of the disease as shown in the annexed table. Recent bacteriological investigation shows that quite a number of organisms are found associated with the disease. Netter[2] gives the bacteriology in 42 cases, — in 25 primary cases Friedländer's coccus or pneumo bacillus was found in 10; streptococcus in 8; staphylococcus in 5; Fränkel's capsuled coccus in 2. In 17 of the cases there was mixed infection: thus in 5 pneumococcus and streptococcus; 5 streptococcus and staphylococcus; 3 streptococcus and capsuled coccus; 2 pneumococcus, streptococcus, and staphylococcus; 1 pneumococcus and capsuled coccus. Similar results were obtained by Durck,[3] who also demonstrated that pneumococcus was the organism most frequently met with. Mosny[4] and Netter both agree that in cases marked by lobular distribution, streptococcus was usually met with alone or with pneumococcus, whereas these in which the distribution was lobar, catarrhal, and fibrinous variety combined, one or other of the forms of pneumococcus, either Fränkel's or Friedlander's organism, were generally found alone. In secondary pneumonias the micro-organisms are still more numerous and varied according to the nature of the primary disease, and associated with the organisms peculiar to the primary disease others, such as streptococci, staphylococcus, and pneumococcus, are met with, showing a

[1] *München. Med. Wchnschr.*, 1895, Bd. xxxii.
[2] *Arch. de Med. nav.*, Paris, Jeurs, 1892.
[3] *Deutsches Arch. f. Klin. Med.*, Leipzig, 1897.
[4] *Thèse de Paris*, 1891.

ACUTE PNEUMONIA

PRIMARY

Catarrhal Pneumonia

- Catarrhal
- Fibrinous and Catarrhal

SECONDARY

Fibrinous Pneumonia

- = Pneumonia = Pleurisy
- = Pneumonia + Pleurisy } Pleuro-pneumonia

DISTRIBUTION

Catarrhal

One or both Lungs
Lobular
Secondary—Primary (more rarely)

Fibrinous

One Lung (generally)
Lobar
Primary

ANATOMY

Catarrhal

Bronchi thickened, exuding pus alveoli, and bronchi filled with desquamated epithelium and leucocytes, sometimes fibrinous material; alveolar septa thickened; surrounding emphysema

Consolidation incomplete
Pleurisy generally present—localised

Fibrinous

Air cells filled with fibrinous exudation containing red and white blood cells and desquamated epithelium

Consolidation complete
Pleurisy present—general

BACTERIOLOGY

Catarrhal

Pneumococcus—mixed infection
Streptococci—Staphylococci—Pfeiffer's bacillus
Measles, Diphtheria, Pertussis
Tubercle Bacillus, Bacterium coli (Sevestre and Lesage)

Fibrinous

Pneumococcus
(Fränkel's—Friedländer's)

mixed infection. Meunier[1] has investigated a large number of influenza pneumonias, in all of which Pfeiffer's bacillus, along with in most cases other organisms, were present. In broncho pneumonias of intestinal origin, both Sevestre and Lesage have demonstrated the presence of the bacterium coli commune. In like manner M. Darier,[2] who has investigated diphtheria pneumonias and many other secondary forms, has found, in the case of diphtheria, Loeffler's bacillus either alone or associated with other organisms. In other diseases, such as pertussis, measles, scarlet fever, erysipelas, and typhoid, similar results have been obtained, and in most of these diseases streptococcus was the predominating organism. Bacteriology thus demonstrates that the pneumonia of infancy can in no sense be considered a specific disease in the sense that it is due to any special organisms, as similar, if not identical, pathological changes are produced in the lung tissues by a variety of organisms. Grancher,[3] in his treatise, says: "The bacteriology of broncho pneumonia is very complex . . . depending on numerous pathogenous organisms, for whereas fibrinous pneumonia is associated almost invariably with pneumococcus, catarrhal pneumonia is produced by a great variety of infections."

Pathological anatomy.—In *fibrinous pneumonia* in the earliest stage there is simple engorgement with congestion of the vascular network of the alveoli, the lung being still vesicular. This soon passes into the stage of solidification, in which the air cells become filled with fibrinous lymph and embryonic epithelial cells from the walls of the alveoli, as well-coloured blood cells and leucocytes. To this stage succeeds that of grey hepatization, the fibrinous network begins to disappear and the leucocytes show signs of commencing fatty degeneration. Fibrinous plural exudation shows itself in the second stage, and is most copious when the third stage is reached and the lung becomes absolutely solid and non-vesicular.

The pathological anatomy of catarrhal pneumonia presents a different picture. The lung is not solid but vesicular throughout. The surface, however, shows isolated lobules or groups of these, prominent and of a purplish colour. Pleurisy is either absent or present, generally in localised areas. On section the

[1] *Arch. Sém. de Méd.*, Paris, 1897.
[2] *Compt. rend. Soc. de biologie*, Paris, 1885.
[3] "Traite des maladies de l'enfance."

lung soon becomes of a scarlet colour, the surface shows irregular small areas of lobular consolidation of a more or less greyish colour, and when the finger is passed over the surface Hamilton describes the feeling like that of a frog's spawn. On squeezing the lung, catarrhal fluid exudes from the smaller bronchi and also from the inflamed lobules. Microscopic examination shows the air cells filled with mucous fluid and cellular elements. These cells are found to be germinating epithelial cells which are rapidly thrown off from those of the alveolar wall and fill up the alveolar spaces. The older epithelial cells desquamate and rapidly undergo fatty degeneration. The whole process is essentially a catarrhal one. While the morbid conditions just briefly alluded to in the two forms are sufficiently distinct, the true fibrinous or lobar consolidation rarely presenting much variation, in the catarrhal variety the appearances are not always of such a typical nature,—in many cases a condition akin to splenization taking place in groups of lobules or larger areas forming more or less solidified lung. On microscopic examination in these cases, the lung shows, in addition to the catarrhal process, more or less exudation of red and white blood cells into the alveoli and interstitial tissue, small blood extravasation being often visible underneath the pleura. In these cases the walls of the minute bronchial ramifications are much thickened and infiltrated, and emphysema surrounding the affected lobules is generally met with to a greater or lesser extent in this form of the disease.

Of 142 cases of acute pneumonia in hospital practice in which recovery took place, 107 showed clinical features of a catarrhal nature, 35 of a fibrinous nature. In 83 of the catarrhal cases the disease ended by lysis, in 24 by crisis. In those of a fibrinous nature—35 in number—1 ended by lysis and 34 by crisis. Both lungs were affected in 53 cases, one lung in 89 cases. In 19 the right apex, in 9 the left apex—28 in all. The base was affected in 27—13 the right base, 14 the left base. Mid lung was affected in 35—22 on the right side, 13 on the left.

Clinical types.—Based on the classification already given, two principal varieties may be noted and each of these may be subdivided. *Primary* acute pneumonia is usually epidemic in its visitation, sporadic cases occurring at times as in all other

infectious diseases. The epidemic nature of the disease is well brought out in hospital practice—groups of cases being met with within short periods, at other times the hospital wards being free from the disease. It is highly probable that the disease is more or less infectious under certain circumstances, but in my hospital experience I have not been able to obtain sufficient evidence to adduce proof of this. During the last six months in the Royal Hospital for Sick Children, we have had a large number of cases, illustrating all the different types from acute infective bronchial catarrh to the fully developed disease, showing either the fibrinous form with lobar distribution or the lobular form more or less distributed through both lungs. With reference to acute bronchial catarrh of the larger and medium tubes, without any clinical evidence of involvement of the alveoli or capillary bronchi, my experience points to the occurrence of these cases more or less frequently during pneumonia epidemics. The clinical features of such cases show sibilant rhonchi and crepitant râles in the larger and medium tubes, with a temperature seldom rising above 103°, the cases generally ending by crisis or rapid lysis within a week or ten days, when the child becomes convalescent and gets rapidly well. None of the cases observed ending fatally and no expectoration being procurable, I have been unable to determine the nature of the organism causing the disease, but the probability of pneumococcus infection seems most likely.

Fibrinous pneumonia.—This form shows two well-marked varieties—one in which the pneumonia is the prominent factor, the other in which pleurisy predominates so as to mask the pneumonia to a great extent (pleuro-pneumonia). In both forms pleurisy is generally distributed, in the first variety, the ordinary lobar pneumonia, that the plural exudate is minus in no way obscures the well-marked typical signs of lobar consolidation. On the other hand, when the fibrinous pleural exudate is very copious, the signs of lung consolidation are less prominent, those of pleural exudation more so, the breath sounds being less distinct and the dulness remaining long after the crisis, the case becoming more or less chronic in its duration and ending by lysis. The occurrence of empyema is a well-known sequel of fibrinous pneumonia in children. Fibrinous pneumonia is a much less fatal disease than catarrhal pneumonia, and is almost invariably primary in its nature. It pursues a well-marked and

typical course, ending in crisis at the end of the week, and is essentially a pneumococcus infection usually attacking previously healthy children. The pulmonary exudate is essentially fibrinous in its nature, and the distribution of the disease lobar and sharply defined in one or other lobe of the lung, contrasting markedly with the diffuse and irregular distribution of the catarrhal variety.

Catarrhal pneumonia or bronchi pneumonia.—The former term seems preferable as indicating the pathological nature of the morbid condition. Whereas fibrinous pneumonia is almost invariably a primary affection, this disease is either primary or secondary, statistics showing that the two varieties are met with in about equal proportions. The primary cases show either a pure pneumococcus infection or several bacteria may be found. The secondary infections are various and generally mixed, streptococci being the most frequent organism. Reference has already been made to the nature of the other infections. This is essentially the pneumonia of infancy, being much commoner at the earlier than the later periods of child life. It is much more fatal than fibrinous pneumonia, and is a disease of indefinite type and duration, coming on insidiously in marked contrast to the sudden onset of fibrinous pneumonia. The pathological changes in the minute bronchi and alveoli are essentially catarrhal, but in a large proportion of cases a fibrinous exudate complicates the catarrhal process. The pathological changes in the lung are irregularly distributed generally in both lungs and the lesion not sharply defined as in the purely fibrinous form, the physical signs of bronchial catarrh being usually present. The duration of the disease is quite indefinite, varying from ten days to many weeks, and resolution is slow, with a tendency to chronicity. Relapses are not infrequent. When the case becomes chronic, interstitial changes take place in the lung, and tuberculosis may ultimately complicate the disease. Pleurisy is generally present to a greater or lesser extent, and is locally rather than generally distributed. Empyema may result from pneumococcus, streptococcus or mixed infection.

Symptoms and physical signs. — *Fibrinous form.* — The clinical features of this form are so well defined and regular and so well known, that I shall only make passing allusion to them, preferring to dwell more fully on those of the catarrhal

varieties as being more interesting and difficult to appreciate. The onset of fibrinous pneumonia is sudden, often ushered in by vomiting in place of rigor, more rarely a convulsion. The temperature soon runs up to 103° or 104°, and remains without much variation till the fifth or seventh day, when a crisis takes place. Should this not occur, it is an indication either of further involvement of the same or opposite lung or some other complication, often extensive pleural effusion (pleuro-pneumonia). The physical signs of this form of pneumonia in a typical case are distinct and constant. The *percussion note* becomes rapidly impaired during the process of consolidation over the affected area, the note over the non-affected portion of lung, and in the opposite lung after showing hyper-resonance. The auscultatory signs are first those indicating pulmonary congestion, high pitched but distant respiratory murmur; sometimes the breathing is so distant as to be scarcely audible. The fine characteristic crepitations heard only at the end of the inspiratory act may or may not be audible. If heard, it may only be detected for a few hours, after which all moist sounds disappear. When consolidation becomes complete, bronchial breathing is audible, as air has ceased to enter the affected portion of lung. The vocal resonance is increased and of a bronchial character. Friction sounds may or may not be heard. As resolution takes place the respiration becomes less bronchial and more vesicular, with coarse crepitant râles (*redux*). Although this regular development of physical signs is the rule, it not unfrequently happens that the signs are delayed, and when detected—perhaps not till the crisis has taken place—are very limited and out of all proportion to the constitutional symptoms, showing that the general pneumococcus infection really constitutes the disease more than the local condition, hence the applicability to this affection of the term pneumonic fever.

Catarrhal form.—This is essentially the pneumonia of infancy. The question why a similar infection should produce in an infant a catarrhal pneumonia and in a child over five years of age or an adult a fibrinous pneumonia, is of much interest. The answer is not far to seek. The infective micro-organism attacks the most vulnerable part of the respiratory tract, which in an infant is the minute bronchi and alveoli of the lung. A little consideration of the developmental processes

going on in the infant lung elucidates this. From the investigations of Nothrup and others, it has been shown that in the late fœtus at five months the alveoli have not yet appeared, the ultimate bronchial ramifications ending in a loose connective tissue which later on becomes thinner but more dense, and in which the vascular network is distributed, and, over this flat epithelium soon becomes developed and can ultimately be differentiated from the columnar variety lining the minute bronchial ramifications. It is not until the fourth or fifth year that the delicate tissues constituting the alveoli of the lung are fully developed. Prior to this time the air cells are much smaller than in the adult, their walls relatively thicker, the interstitial tissue being larger in amount. The blood-vessels of the alveolar walls are likewise very abundant. The connective tissue cells in the stroma and also the epithelial cells are very numerous and readily proliferate. Such being the anatomical condition of the infantile lung, it becomes evident that a ready explanation is afforded of the pathological conditions produced by disease in these delicate growing tissues: the greater vascularity of the walls of the alveoli and the abundance of their epithelial contents, the immature nature of the cells, and also of the cellular elements of the interstitial connective tissue, forming a group of anatomical conditions possessing less immunity to the infective process than any other portion of the lung tissue.

Symptoms and physical signs.—These form a marked contrast to the constancy and regularity of fibrinous pneumonia pure and simple. Grancher[1] introduces his description of the clinical picture of this form of the disease by generalising its features by his very apt terms "multiplicité, complexité, mobilité." In order to appreciate the varied nature of the clinical characters of the disease, it is necessary to premise that there are three pretty distinct varieties: (*a*) Those without signs of consolidation, (*b*) with small areas of consolidation (lobular distribution), (*c*) large areas of consolidation (lobar distribution).

I shall first refer to symptoms and then describe shortly the physical signs by which we are aided in recognising the nature of the disease.

Symptoms.—These are much more constant than the

[1] "Traite des maladies de l'enfance."

physical signs and show a marked similarity in all the varieties and a common ground enabling the physician with tolerable certainty to arrive at a diagnosis without the aid of physical signs. When the child is stripped and inspected it is noticed that there is more or less respiratory embarrassment and probably cyanosis, the alæ nasi are in active motion and the respiratory movements altered, showing more movement of the chest walls than normal in the young child, with more or less inspiratory retraction of the lateral basis and diaphragmatic insertion. The pulse respiration ratio is perverted, the respirations running up to 60 or 80 per minute. The temperature ranges from 102° to 104° or higher, and is of an irregular type. With such a group of symptoms you may predict with tolerable certainty that the auscultatory signs will reveal the presence of catarrhal pneumonia.

Physical signs.—These vary infinitely according to the physical condition of the lung. It may be at once stated that auscultation is the means by far the most reliable of diagnosing the pneumonic areas, percussion, except in large areas, being unreliable for various reasons hereafter to be noted.

It is well known to pediatricians that some of the most severe and dangerous cases of catarrhal pneumonia, such as those seen after measles or in acute tubercular infection, present no physical signs of consolidation of the lung. Clinically, these cases may be divided into two varieties, one to which I have already alluded, a pneumococcus or mixed infection of the larger and medium tubes (not a true catarrhal pneumonia as the alveoli are not implicated) which run an acute course ending in crisis or rapid lysis. In these cases the physical signs are these of ordinary bronchitis and need not be further alluded to. The ordinary form in which all the branches of the bronchial tree are involved, as well as the alveoli, may be either primary or secondary. Here the temperature chart exhibits the usual type of acute catarrhal pneumonia, with the rapid respiration and cyanosis. The percussion is generally resonant. The auscultatory signs are sibilant rhonchi, with coarse and fine crepitant râles, more or less distributed over one or both lungs. Over certain areas of the lung where the alveoli are most affected finer râles are heard in corresponding portions of lung. These râles are highly characteristic to the practised ear, being

audible chiefly during the whole of the inspiratory act, but frequently during expiration as well. They take their origin in the minutest bronchial ramifications and alveoli. The general character of the respiration in these cases is vesicular, but owing to the congested condition of the lung the breathing is feeble, then normal.

The *second variety*, when the distribution of the pneumonic areas is lobular, is characterised by signs indicating localised consolidation in patches varying in size. Even in this variety the information gained by percussion is often unreliable. Auscultation on the other hand soon detects the site of the affected portions of the lung. In using the stethoscope in these cases, every inch of ground, so to speak, must be gone over in order to detect the locality of the pneumonic area. Sometimes these are so small as to be included only by the end of the stethoscope; if the instrument be moved away from the spot the characteristic signs are lost. These small pneumonic areas tend to enlarge and coalesce with other and neighbouring patches so as to form larger consolidations over which the physical signs become more and more distinct. The percussion note is more or less impaired over the affected portions in proportion to the size of the areas involved and their proximity to the surface. In some cases there is little or no impairment of the percussion note owing to the surrounding healthy lung tissue, and if there be much emphysema the note may be even hyper-resonant in places. It will thus be evident that in discovering these small lobular distributions of affected lung the stethoscope must mainly be relied on. It is often difficult to impress on students, in discussing this form of pneumonia, the necessity of eliminating dulness on percussion as a reliable or constant physical sign. The auscultatory sign over the affected portions are broncho-vesicular breathing with increased crying resonance and moist sounds of the same character as those described in the former variety, with this difference, that sometimes over the centre of the affected lobular areas there are few or no râles, the accompaniments being more numerous and distinct towards the peripheral parts. The vocal fremitus is either unaltered or slightly increased.

In the *third variety*, with larger areas of affected lung and lobar involvement, the physical signs are more distinct and

easily appreciated. In many cases there may be some difficulty by physical signs alone in distinguishing the consolidation from a purely fibrinous one, but taking into account the general features of the case otherwise, the presence of bronchial catarrh, the incompleteness of the consolidation (for it must be remembered that in catarrhal pneumonia the alveoli contain more or less air), the distinction may easily be drawn. In the lobar consolidation of catarrhal pneumonia the dulness gradually shades off and is not so sharply defined as fibrinous pneumonia. Crepitant râles are usually present during the entire course of the disease, and the dulness or percussion is less marked—in fact it is usually very incomplete in proportion to the extent of the auscultatory signs.

Enough has been said to show the extreme variations in the physical signs in catarrhal pneumonia; but taken along with the general symptoms there is little real difficulty in determining the true nature of the case.

Before I refer shortly to the treatment of acute pneumonia, a word may be said as to the terminations of the disease. In the fibrinous form recovery takes place in about 95 per cent. of the cases; in the catarrhal form the mortality averages probably about 60 per cent., this being largely due to the fact that this is the form which attacks children under two years of age, that a large proportion are secondary cases, and that complications are more numerous, and the tendency to chronicity and ulterior destructive changes in the lungs frequent. When this form of the disease becomes chronic, tubercular infection is also apt to occur. The destructive changes in chronic broncho pneumonia are generally of an interstitial nature ending in fibroid changes. Empyema, although not so frequently met with as in the fibrinous form, is by no means uncommon. It is most frequently of a localised nature, sometimes very limited, only containing a few drachms of pus. Such small accumulations when detected early are easily and successfully treated by aspiration alone. The most troublesome and frequent complication of broncho pneumonia is intestinal catarrh, which often proves troublesome and intractable.

Treatment.—As yet we have no special treatment for acute pneumonia. No anti-toxin has yet been discovered, but this is a line of research well worthy of attention. All we can do is, treat each patient according to the special needs of the case,

meeting symptoms as they arise, by rational means. In all cases hygienic measures, including diet, are all-important. The patient should be in a well-ventilated room of mean temperature. I find a tent bed invaluable, hung round inside with wet towels sprinkled with eucalyptus or creasote or other antiseptics. In very severe cases, with much cyanosis, the inhalation of oxygen mixed in the air of the tent often proves of signal benefit. The dieting should be conducted on the general principles applicable to all febrile affections : light, easily digested nutriment in such quantity as the patient can digest. Over-feeding is harmful as likely to bring on vomiting and diarrhœa, which are complications always to be guarded against. Continuous and routine poulticing is now discarded, poultices being used as other therapeutic means, according to the necessities of the case. A stimulating sinapism, occasionally applied all over the chest for half an hour or an hour, is often very serviceable, this being replaced by a pneumonia jacket of Samger tissue. When the temperature is high and the skin is not acting, a moist continuous poultice of soft flannel and jaconette, moistened with boracic solution, is very grateful to the patient. By keeping up a local as well as general diaphoresis, relief is given to the engorged lung whose blood-vessels are in a state of hyperæmia and permanent dilatation, the cutaneous blood-vessels which are in the opposite condition of high tension, being thereby relieved.

Drugs treatment.—As no drug yet known has any special influence on the disease, we must treat the patient by endeavouring to assist Nature in her efforts to maintain a compensating physiological balance in the functional activity of the various organs. In the reference already made in regard to dieting, I have indicated the necessity of due attention to the *digestive functions.* The respiratory embarrassment can be best relieved by keeping up a continuous local and general diaphoresis as already indicated. This should be assisted by the exhibition of non-depressing diaphoretics such as liq. acet. ammonii and sp. ætheris nitrosi, a combination which has well stood the test of experience. The nitrous ether is most valuable in relieving the general vascular tension apart from that of the lung. Alcoholic stimulants are of the greatest value, especially in broncho pneumonia. They must be administered in suitable doses, according to the exigencies of the case and the state of the pulse.

Next in importance as a stimulant I regard strychnine. The indications for its use are similar to those of alcohol, and it may be given with advantage at the same time. I regard it as of more value in the acute respiratory affections of children than in adults, on account of its action on the cardiac and respiratory centres and cardiac ganglia. In children it is well known that the nerve tone is more rapidly lowered from acute disease than in adults, hence the great importance of anticipating this by the timely use of such a remedy.

The use of belladonna in certain cases of acute respiratory affection of infants is one of the most striking therapeutic facts I know of. The cases in which it is indicated are those particularly of infants attacked with acute congestive bronchial catarrh in the early stages. It is well known that in these cases there is great respiratory embarrassment due partly to the congestive condition, which is accompanied by more or less bronchial spasm and collapse of the lung. Its action in soothing the afferent and efferent nerves in the bronchial walls and stimulating the respiratory centre is most marked. It has no beneficial effect in the later stages when the alveoli are involved, but its timely use in the earlier stages of the disease is attended with striking results.

My remarks on the drug treatment of the acute pneumonias would be incomplete were I not to refer to digitalis, and I only do so to condemn its use as a *routine* remedy. I do not for a moment undervalue the importance of employing this drug in cases where its use is clearly indicated—feeble pulse with weakness of the left ventricle and diminished arterial tension. Such conditions obtain occasionally, but not frequently, in the advanced stages of acute catarrhal pneumonia. In fibrinous pneumonia with large lobar consolidation, on the other hand, the condition of the arterial system clearly contraindicates its use. The condition of the blood-vessels in the affected part are those of distension and paralysis, the systemic arterial circulation being in a high state of tension, with an enfeebled and distended right heart. In such cases we have no proof that digitalis has any power in restoring the tone of the blood-vessels in the affected part. It cannot act on the right heart without more powerfully, on account of the greater muscular area, stimulating the left ventricle to congest the affected lung by "diminishing the vascular area" (Loomis).

Heart failure in lobar pneumonia, as I indicated in an early part of this paper, would appear to be due to two principal causes—the depressing effect of the toxines on the nervous centre, and of a high temperature on the heart muscle. If this be so, there are no indications for digitalis, but rather for such remedies as act directly by stimulating the nerve centre on the one hand, and relieving the hyperæmia on the other. I believe that in local and general diaphoresis, occasional purgation and remedies which stimulate the cardiac respiratory centres are the appropriate means of combating this condition.

Anti-pyretics.—The use of large doses of phenacetin or antipyrine are, in my experience, not useful, but often harmful in both forms of pneumonia. In small doses, however, they are often of service in allaying nervous instability and restlessness. In hyper-pyrexia we must trust to the wet pack, or tepid bath, or the application of cold by ice bags or otherwise.

Expectorants.—I regret to find that even yet in these more enlightened days such remedies as ipecacuanha, tartar emetic, and squills are still prescribed by some physicians. There is no indication that I know of, but a distinct contra-indication for their use. Children suffering from pneumonia require rather stimulation than the exhibition of depressing remedies such as these.

2. A FIT OF GOUT (PAROXYSMUS PODAGRÆ): A STUDY IN PATHOLOGY

By GEORGE W. BALFOUR, M.D., LL.D.Edin. and St And., F.R.C.P.Ed.

"Non fingendum, aut excogitandum, sed inveniendum quid Natura faciat, aut ferat."—BACON.

GOUT as a disease has been known to physicians from time immemorial, yet its most graphic description is of no earlier date than Sydenham, who, in memorable words, has depicted it with all the skill of a physician and all the accuracy of a sufferer. Succeeding writers have but copied Sydenham's delineation of the disease, and have modified his theory of its rationale. This, as we know, was a reversion from the doctrine prevalent in his day, of tartar accumulated in the blood and deposited as chalk stones in the joints, to the still older theory of an effort of the system to expel a peccant humour, the result of imperfect coction. To Cullen's logical mind we owe the

differentiation of gout into its several varieties of regular, atonic, and aberrant. And to Cullen also is due the statement that the supposition of any morbific matter is unsupported by any evidence, is quite superfluous, and is useless, "as it has not suggested any successful method of cure."[1] Cullen's own idea was that gout "is manifestly an affection of the nervous system."[2] Modern advances in zoo-chemistry and in morbid anatomy have introduced various modifications of our ideas as to the ultimate pathology of gout. Some of these modifications are more closely allied to the humoralism (Haig, etc.) and others to the solidism (Ord, Bristowe, etc.) of our forefathers; while even those who with Cullen regard the nervous system as the *primum mobile* of this disease (Duckworth, etc.) are forced to recognise the exciting influence of a pre-existing toxæmia.

With all our increased accuracy in details, it does not appear that our ideas of what gout really is are any clearer or any better defined than those of our forefathers. To the non-professional mind gout presents itself in two aspects—first, as an embodiment of ill-temper, an agonising toe, somebody's specific, and short commons; and, second, as a meaningless epithet, vaguely applied by physicians to all ailments occurring after middle life which are obscure in their origin, variable in their symptoms, and not readily amenable to treatment. As to the professional idea of gout I speak with all humility, having been unable with any certainty to discover what is the prevalent professional conception of this disease; but I presume, subject to correction, that the clearest heads among us regard it as a toxæmia with a uric acid basis, the nervous system as a ruling spirit, and the whole organism as a *campus martius* wherein to exercise. What idea our younger brethren entertain of gout it would probably be hard to imagine, but it must seem to them as a most mysterious complication of life, when they are taught that not only may it hop like a bird from one part of the body to another, but also that such very tangible affections as "hæmatemesis and hæmoptysis are often cured by a regular fit of the disease" (Gairdner). A former member of this society, Dr Craigie, in his "Practice of Physic," has said that it is in the natural history of gout that its real character and nature is to be sought; and this idea has been repeated and emphasised by the late Dr Todd in his

[1] *First Lines*, ccccxcii. [2] *Ibid.*, ccccxciv.

"Croonian Lectures." This view I myself homologate, and I am persuaded that a clear idea of the true nature of a paroxysm holds the clue to much that seems mysterious in the history of gout, and puts into our hands the means of preventing, if it cannot always enable us with any certainty to cure, its manifestations. The natural history of a paroxysm may not explain all the mysteries of gout, but it gives us a clue that will suffice to guide us through the labyrinth of symptoms presented by this protean malady.

I cannot, I regret to say, show you an illustration of a gouty toe in its paroxysmal condition. So far as I know, no such delineation exists; and he would be a bold artist, indeed, who should propose to sketch such a toe from nature. A mere fancy sketch would be of no value for my purpose, so I must ask you to recall the simulacrum of such a toe if you have ever seen one; and, if you have not, you may still, I hope, be able intelligently to follow me as I point out and endeavour to explain the rationale of the several signs described by Sydenham and others.

Podagra, you may remember, is the name given by Cullen to the twenty-third genus of the order Phlegmasiæ, class Pyrexiæ. Cullen adopted the name Podagra from Boerhaave and the older writers, because it correctly, if somewhat vaguely, described the great characteristic of the disease (a foot seizure) without committing him to any definite theory of its nature, explicitly rejecting the term Arthritis as misleading, and as conveying an idea (of inflammation) for which there is absolutely no foundation. Podagra is distinctly a febrile (pyrexial) disease; it has also all the characteristics of a phlegmasia, but of the three characteristics by which an external inflammation is recognised—*calor*, *rubor*, *et tensio dolens*, the first is conspicuous by its absence. Moreover, Podagra invariably terminates by resolution, and never ends, as inflammations so often do, either in suppuration or in gangrene.

Sydenham tells us that on the morning after the commencement of an attack, the part affected—the metatarsophalangeal joint of the great toe—is found to be swollen, but that up till this time "the only visible swelling had been that of the veins of the affected joint."[1] Turgidity of the veins coming from the affected joint is thus the earliest visible sign

[1] *Syd. Soc. Translation*, p. 125.

of an attack of gout, and this sign persists till convalescence sets in. But venous turgescence is not an accompaniment of inflammation. The veins of an inflamed part undoubtedly carry a larger amount of blood than in health, because the dilated arteries of an inflamed part convey to it a larger amount of blood than usual, and this has to be returned by the veins, but these vessels remain inconspicuous and unnoted amid the general turgidity of the part. In the case of a paronychia, for example, which is an inflammatory affection of the distal extremity of a digit, and in situation, at least, closely resembles a fit of gout, there is redness, heat, and painful swelling, but the veins are neither turgid nor visible. The dilated arteries carry an excess of blood to the part, but the blood path is everywhere free, and the blood is speedily restored through the veins to the general circulation. Should, however, the blood path be choked by an embolus or a thrombus, the case is altogether different. When such a block occurs, in a vessel with many collateral communications, the circulation is scarcely checked and is speedily restored to its normal. But should the block occur in a terminal artery, an anæmic area is at once formed in front of and around the obstruction. When the circulation around this area is carried on through capillaries and small arterioles, no true infarction is formed ; but from the sluggish movement of the blood within the anæmic area a stasis occurs in the vessels, which, if visible at all, is most evident in the veins leading from the part up as far as a more or less distant point where they enter a sphere in which normal conditions prevail. Within the anæmic area the augmented tension and diminished vitality of the walls of the vessels, due to the sluggish flow of the blood, cause an accumulation of plasma in the part, accompanied by a diapedesis of the corpuscular elements. Hence there is swelling of this area ; and as the red corpuscles part with their colouring matter to the plasma, the swelling is of a dusky redness, which gradually deepens till the normal conditions are restored.[1]

Now, if we turn to a gouty paroxysm, and limit our consideration to its essential characteristics, we find that it commences with a sudden attack of agonising pain in the part

[1] *Vide* paper by Dr M. Litten in the *Ztschr. f. klin. Med.*, Berlin, Bd. i. S. 189 ; and Cohnheim's "Lectures on Pathology," *New Syd. Soc. Translation*, vol. i. p. 121.

affected—the metatarso-phalangeal joint of the great toe. But pain is not an exalted condition of nerve function. As Anstie has said, pain is the prayer of the nerve for more blood, for better nourishment. Whatever interferes suddenly and more or less completely with nerve metabolism gives rise to intense pain. Hence the excruciating agony that follows the occlusion of a vessel, whether artificially, as in the treatment of aneurysm by compression ; or naturally, as in gangrene following blocking of an artery by embolism or thrombosis, or in many other similar processes. In a gouty paroxysm the excruciating agony comes on suddenly, and is perfectly explicable, on the theory of thrombosis of the small vessels of the joint affected. Moreover, the sequential phenomena are precisely those that would follow the formation of an anæmic area in and around the joint implicated. First of all, before there is much if any manifestation of local swelling,[1] there is visible turgidity of the veins leading from the affected joint up to the dorsum of the foot, till they merge into a sphere where normal conditions prevail. Next, we have swelling in and around the joint, from accumulation of plasma within the anæmic area. And, lastly, this swelling becomes increasingly tense, stretching the skin and giving it a glistening appearance, which is perhaps all the more striking from the deepening dusky redness due to the solution of the colouring matter of the red corpuscles of the plasma.

All the events of a gouty paroxysm are thus readily explicable, on the theory of thrombosis of the small vessels of the joint affected, and upon no other theory with which I am acquainted ; and all the concomitant phenomena are corroborative of this view.

For example, a gouty paroxysm is commonly ushered in with a rigor followed by a rise of temperature—phenomena of daily occurrence in connection with embolism—with sudden blocking of the circulation. The affected joint presents, we know, many of the indications of inflammation, the *rubor* and the *tensio dolens* are very much in evidence, but the *calor* is conspicuously absent. I have occasionally taken the temperature of the distal extremity of a gouty toe, and found it to be invariably subnormal. Sir Dyce Duckworth[2] tells us the same story. He says he has repeatedly taken the temperature of a

[1] Sydenham, *op. cit.*, p. 125.

[2] *A Treatise on Gout*, p. 248. The bodily temperature in gout is seldom over 102°, but has been known to rise to 104°.

gouty toe by putting a thermometer close to it and keeping the two in contact for twenty minutes with a roll of cotton wool,[1] and has always found it subnormal. On two occasions it was 97° and 95·5°, the corresponding temperatures in the mouth being 100° and 100·4°. I am aware that the temperature of the corresponding toe on the opposite foot has also been found subnormal, but, however we may explain this,[2] it does not lessen the remarkable fact of the distal part of the affected toe which is tumid, red, and painful, and to the sufferer feels hot, having a temperature two or three degrees below that of the body generally; a state of matters which seems wholly inexplicable on any other theory than that of a stasis of the circulation at the part affected.

After a fit of gout the tissues about the joint and its cartilages are found to be infiltrated with urate of soda, a condition trifling after a first attack, but which increases with each succeeding paroxysm. This condition Garrod regards as of the highest importance as indicating a specific form of inflammation,[3] a form of inflammation which would indeed be not only specific but peculiar and altogether *sui generis*. On the other hand, we know from the researches of Garrod himself that in gout the blood serum contains uric acid,[4] and that this is also found in the serous fluid within the pericardium, the peritoneum, and the subarachnoid space of gouty subjects.[5] The concretions of urate of soda upon the auricular cartilages and on other parts, and the nodosities about the digital joints, known as gouty pearls and Heberden's knobs, are not preceded by any inflammation, but probably follow some slight local injury. At first —if we see them early enough, which is rarely the case—we find a small localised and, as it were, encysted effusion, within which the urate of soda slowly crystallises, the remaining serum is absorbed, and the pearl or knob is left behind. The so-called pearls rarely grow, generally indeed become harder and smaller;

[1] This method of taking the temperature makes it absolutely certain that it was subnormal, as by covering the bulb of a thermometer with cotton wool we may so prevent the dissipation and favour the accumulation of heat as apparently to raise the perfectly normal temperature of either the mouth or the axilla up to 108° or even higher.

[2] The low temperature of the opposite foot may be due to reflex contraction of its arteries from the intense pain of the podagra. Just as Broun-Sequard and Tholozan found that pinching raised the temperature of the limb pinched, but lowered that of the opposite one.—*Gaz. méd. de Paris*, 1870, p. 142.

[3] Third edition, p. 187. [4] Third edition, p. 84. [5] Third edition, p. 109.

but the knobs, by a repetition of the process, do grow, and more and more disfigure the extremities.

Urate of soda is a somewhat insoluble salt, always present in the blood serum of gouty patients; and when the plasma accumulates within the anæmic area of a gouty toe, the salt slowly crystallises out of the extravascular fluid, and gets left behind, infiltrating the cartilages, the cavity of the joint, and the tissues surrounding it, when the serous part of the effusion gets reabsorbed, as the circulation returns to its normal. Each returning paroxysm increases the amount of these extravascular urates, and also the disfigurement of the joint; but their deposit is not the result of any inflammatory action, nor is their presence in the joint ever the cause of any inflammation, beyond the disfigurement; their presence in and around the joint seems to give rise to no disturbance and to be absolutely neutral, so far as the state of the person so disfigured is concerned.

There is nothing, therefore, in the history of a gouty paroxysm which connects it with any form of inflammation, and all the phenomena present are readily—and seemingly only—explicable on the theory of thrombosis of the small vessels of the joint affected, and the formation of an anæmic area around their sphere.

Nay, more, the mode of prophylaxis, which has commended itself to many sufferers as a certain preservative from all the ills of an acute attack, as well as one method of cure which, in the hands of a bold practitioner, has proved eminently successful, are not to be explained, so far as I can see, upon any other theory whatever.

Cullen narrates some curious instances of the sudden cure of gout. One man, long confined to bed with gout, when his neighbour's house took fire, ran off as fast as he could. The provoked antagonist of another patient, dressed up as a spectre, and throwing the sufferer over his shoulder, dragged him downstairs, bumping his poor feet against every step; at the bottom of the stair the ill-used man found his legs, and ran upstairs fast enough. While a third, on the first premonition of an attack, took his dog and his gun, and walked it off.[1] Sir William Temple tells us of the Rhyngrave, who was killed before Maestricht in 1676, and was a friend of his own; on

[1] *First Lines*, 2nd edition, 1778-84, MS. notes, p. 389, etc.

the first indication of a paroxysm, this nobleman went out immediately, whatever the weather, and walked as long as he was able, "pressing most upon the foot that threatened him; when he came home, he went to a warm bed, and was rubbed very well, and chiefly upon the place where the pain began." If the attack continued or returned next day, the same course was pursued, so that he was never laid up with gout; and he thought so well of his treatment that he strongly recommended it to his son, should he ever be attacked in the same manner. Temple also tells us of one of his brother's gamekeepers very subject to gout, but who never laid himself up. When attacked, he busied himself with his deer or his stud, walking about from morning to night in spite of the pain.[1] And the late Dr William Gairdner of London was acquainted with an active, lively old gentleman of eighty-five, who, when seized with gout, always replied to the remonstrances of his family and his physicians, "I'll walk it off," quaintly adding, "Go to bed with the gout, and it will surely go to bed with you, and be mighty bad company."[2] A remark that recalls the statement by Sir William Temple, that gout haunts chiefly those who treat it as a friend, and not as an enemy, who "carry it to bed with them, and keep it soft and warm, and indeed lay up the gout for two or three months, while they give out that the gout lays up them."[3]

These are sufficiently remarkable statements, based upon equally remarkable facts, yet we are told that the idea of attempting to abort a fit of gout could not occur to anyone "who regards it from a proper pathological standpoint."[4] To myself it seems quite the other way about, and that it is only to those who have a correct idea of the pathology of gout that it could legitimately occur to attempt to abort an attack. We are told that "the risks of suppression of local symptoms are so great, and the benefit, if there be any, so dearly bought in most cases, that an ectrotic treatment can seldom be advisable."[5] But we never hear of any evil results following the successful aborting of a fit by active exercise; Dr Gairdner's old friend carried it out to the

[1] *The Works of Sir William Temple, Bart.*, Edinburgh, 1754, vol. ii. p. 127.
[2] Gairdner, *On Gout*, London, 1849, p. 114.
[3] Temple, *op. cit.*, p. 128.
[4] Duckworth, *op. cit.*, p. 348.
[5] Duckworth, *loc. cit.*

end of his life. A friend of Mr Apperly (the Nimrod of old times), after a skinful of wine and other good things, always finished the evening by walking off the soles of his pumps before retiring to bed, in order to ward off gout, and this he did successfully and without damage to the end of his days. Cullen, it is true, tells us of one man who, after warding off many attacks, was suddenly laid on his back by a severe fit,[1] but his failure to check this one could scarcely be attributed to his having successfully overcome previous attacks. Both Cullen and Duckworth, regarding gout as largely an affection of the nervous system, look upon the good results that follow this method of fighting off a fit as an example of the curative effect of "great mental emotion,"[2] or, as an instance, of the "remarkable influence exerted by a vigorous nervous system on the peculiar phenomena of a gouty fit."[3] But no mental emotion, however great, nor any power of will, exerted even by the most vigorous nervous system, can produce any effect upon a gouty digit, unless it be accompanied by movements of the joint sufficiently energetic to dislodge any thrombi, break up the stasis, and restore the circulation in and around the joint affected. On the other hand, we know that manipulation and friction, carried out with or without the will of the patient, are sufficient to restore the circulation and to cut short an attack without any ulterior bad result. It is true we hear but little of this method of treatment in medical literature, yet it must have been occasionally employed, as when Boerhaave, in his 1275th aphorism, under section 8, speaks of curing gout "*exercitio magno, continuato, equitationis in aere puro, tum frictionibus. motibusque partium saepe iteratis*,"[4] the context shows that he speaks of these as approved methods to employ in the cure of an acute attack, and not merely as means to be used for the removal of the rigidity of gouty limbs during intervals of comparative health.

It may be readily understood that, however efficient this method of treatment may have proved itself to be, it is not likely to have been often or lightly resorted to. When the trifling succession caused by merely crossing the floor to the bedside is liable to provoke intolerable agony, he must be a bold

[1] *Op. cit.*, p. 392, MS. note.

[2] Cullen, *op. cit.*, p. 389.

[3] Duckworth, *op. cit.*, p. 349.

[4] Boerhaave, *Aphorismi de cognoscendis et curandis mortis*, Lugdun, Batavorum, 1727, p. 212.

practitioner who would venture to propose to apply massage to the offending digit itself. Yet this has not only been proposed, but also successfully carried out with the most astonishing results.

In the first half of this century we had in Edinburgh a well-known practitioner—Dr William Balfour[1]—who had a great reputation for his successful treatment of rheumatic affections, by the various methods we now comprise under the term massage. Occasionally he attacked more acute ailments with a boldness and a success which were truly remarkable. In the *Edinburgh Medical and Surgical Journal* for October 1816,[2] Dr Balfour relates several cases of acute gout, treated by compression, friction, and percussion. These cases are all remarkable, and all perfectly successful. I shall quote only one, as that is sufficient to show his method and its results.

Case

Mr J. O., solicitor, æt. 47, of a cholerico-sanguine temperament and full habit of body, woke in the morning of 22nd July with violent pain in the balls of both great toes, particularly at the flexures of the joints. "When I visited him at eleven o'clock forenoon, the parts were swelled, red, tense, and shining. The inflammation and pain were confined almost exclusively to the balls of the great toes, except that in the left, which was by much the worst, the pain reached upwards about half way along the metatarsal bone. The patient experienced continual excruciating pain, even when at perfect rest; his countenance indicated considerable distress, and he complained of sickness, general uneasiness, and oppression; pulse, 82.

"When I entered my patient's room, he told me that, if I

[1] William Balfour was the younger son of Alexander Balfour of Tillery, Kinrossshire, where he was born in 1769. He graduated at the University of Edinburgh in 1814, and shortly afterwards, as is stated in an obituary notice in the *Lancet* of 9th May 1846, he "commenced a career which, under whatever aspect it is viewed, has seldom been exampled in the medical world." His paper entitled *Observations on Adhesion, with two Cases demonstrative of the Powers of Nature to restore parts which have been by Accident totally separated from the Animal System*, was both original and acute, and distinguished by perspicuous and forcible language, qualities by which his style was uniformly distinguished. This paper was transferred to every scientific periodical of repute, and at once made him famous. He subsequently turned his attention to the treatment of rheumatism and gout, and speedily acquired a large and lucrative practice. He died in 1846 at the age of 77.

[2] P. 432, *Observations, with Cases, illustrative of a New, Simple, and Expeditious Mode of Curing Gout.*

intended to apply friction to the parts affected, he was afraid he could not suffer it; for he had attempted that himself, but might as well have applied 'living fire.' I told him I knew that friction was quite inapplicable to his case as it then stood, but that I would, notwithstanding, employ mechanical means for moderating the paroxysm, and, I hoped, with complete success. I therefore grasped the ball of the toe in my hand, applying pressure gradually, to a very considerable degree. Slight pressure gave pain; heavy pressure none. In this manner I compressed the parts, sometimes with one hand, at other times betwixt both hands, for about ten minutes, now and then interposing gentle percussion; and then applied a thin flannel bandage. Ordered a brisk purgative of senna and sulphate of magnesia.

"In the evening the patient said he 'had considerably less pain, or rather none at all, from the moment I left him'; but he had a good deal of cold shivering during the day, his pulse was 100. The swelling and redness of the parts had much abated, and motion of the affected joints was practicable. Compression and percussion were reapplied, and were much better borne than in the earlier part of the day. Next morning the patient was found to have slept all night without interruption; his pulse was 80; pain, swelling, and redness of the parts almost entirely gone, and he could suffer them to be handled with the utmost freedom; he walks very well. Compression and percussion were applied in the morning, in the evening, and again next morning, after which the patient went abroad in the prosecution of his business—on the third day of his attack."

Such a case—even if it were a solitary one—would be truly remarkable, but as one of a series it is more than remarkable, it is instructive. The case and its cure are so graphically detailed as to leave no doubt as to the nature of the one or the reality of the other. Dr Balfour points out that, as the roll of the waves does not at once cease when the gale drops, so also the assuaging of the local ill was not at once followed by the cessation of the symptomatic fever, which indeed increased for the first few hours, but speedily thereafter passed away. The general oppression, the sickness, and the dyspeptic symptoms were also relieved, though this, as

the doctor wisely remarks, was no doubt largely due to the purgative remedies employed, and not altogether to the cure of the joint affection; the latter he rightly ascribes to the restoration of the circulation in the part affected.

As we have seen that all the phenomena—objective as well as subjective—of a gouty paroxysm are apparently only explicable on the theory of thrombosis of the small vessels of the part affected, so now we find that a gouty paroxysm can be readily and rapidly cured by measures which can only act by restoring the circulation through the part affected, and which are well calculated to effect this result. But, however delightful it must be to be cured of a bad fit of gout by one or two séances of a practised masseur, I fear there would be a difficulty in getting anyone to submit to an operation which seems to bring with it the possibility of such agony. And I am equally persuaded that there are few, if any, who possess sufficient nerve and skill to carry out effectively so delicate an operation.

On a future occasion I hope to return to the subject, to point out the rôle of thrombosis in relation to all those various affections we term gouty, to endeavour to expiscate the causes of thrombosis in gouty subjects, and to show how they may best be warded off; at present I must content myself with having directed your attention to what seems to be the true cause of a gouty paroxysm, which, as Craigie and Todd have told us, is the first step to a clear understanding of the true nature of gout itself. And I cannot conclude better than in the words of Sir William Temple, who quotes from a Dutch friend who had long lived in the East Indies, that there "the general remedy of all that were subject to the gout was rubbing with hands; and that whosoever had slaves enough to do that constantly every day, and relieve one another by turns, till the motion raised a violent heat about the joints where it was chiefly used, was never troubled much or laid up by that disease."[1] I can so far corroborate this that the few cases I have persuaded to employ massage of the extremities night and morning, have since then kept free from any severe attack.

[1] *Op. cit.*, vol. ii. p. 127.

Meeting VIII.—May 18, 1898

Dr JAMES CARMICHAEL, *Vice-President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Sir Thomas Grainger Stewart* exhibited a CASE OF PECULIAR NERVOUS AFFECTION, showing some analogy with Thomsen's disease.

A. H., aged 64, married; family, three children.

Occupation.—Makes wire frames for working carpet patterns on.

Admitted.—April 18th, 1898.

Examined.—April 19th, 1898.

Complaint.—Difficulty in walking.

Duration.—Six months.

History.—Family history, nothing of note. No other members affected in the same way. Comfortable home. Stands at his work. Good food. Never drank to excess. Smokes 1½ oz. a week (black twist).

Previous illnesses.—Patient does not remember having ever been ill. No history of syphilis.

Present illness.—Began six months ago, when patient was digging in his garden. He found a difficulty when inserting the spade into the ground. He felt his foot trembling. This passed off in a few minutes. When walking the same evening, he noticed that he walked with difficulty, and the muscles of his legs felt stiff.

The difficulty in walking gradually increased, and was especially marked in the mornings and after he had been sitting some time; also when turning a corner. His legs tremble when he attempts to start, and patient attributes his inability to start to this fact. He has never felt giddy, has never had any pain or discomfort, but finds that he is easily tired.

He stopped work a month ago on account of the difficulty he had in going to and coming from work.

State on admission.—Patient is a well-developed man, very untidy, and has a habit of winking.

When starting to walk, he stands with one hand in his pocket, and often scratches his head with the other.

When he stands up and tries to start walking, his legs begin to tremble and his muscles become rigid.

He shuffles for a few inches to the right and a few inches backwards; he then balances himself on his right leg, and advances his left like a person walking on thin ice which he thinks will give way if he advances further. Patient sometimes takes half a minute or longer to start, his legs trembling and his muscles rigid during that time. He will walk two steps, stop and stammer, and then start again. He may then walk for some distance in a perfectly natural manner, or he may fail to start, in which case he will stand and scratch his head or stroke his beard. At the same time there is a semi-serious, semi-comic expression. After a series of attempts he gets breathless, and perspires freely about the forehead.

The only remark he makes is, "I maun wait till I get a start," and, when started, "I'm awa' noo."

There is no lurching to either side when he walks; no giddiness; and the ground feels normal.

There is no indication that the patient is humbugging. He was treated at home by his own doctor for six months before admission, and has never been in hospital before. He nearly cried on several occasions when he found the difficulty in walking greater than usual.

The fact that a big hat pin was driven into his buttocks and thighs made not the slightest difference to his walking.

The various systems are practically normal.

Nervous system.—Sensory functions normal; optic discs normal; no diminution in field of vision.

Motor functions.—Reaction to faradic current slightly sluggish, otherwise normal.

Cerebral and mental functions.—Speech slow; no headaches; sleeps well.

Locomotory system.—Muscles well developed, especially of lower limbs, although not excessively developed. Contraction of muscles when attempting to walk as already noted.

2. *Mr Alexis Thomson* exhibited a CASE OF COMPOUND DEPRESSED FRACTURE OF THE SKULL WITH LOCALISED CORTICAL SYMPTOMS. The patient, a man of 37, was, on the morning of the 6th April, struck on the left side of the head and body by a runaway bogey and by the stones falling from

it. Was unconscious for half an hour. On admission to Dr MacGillivray's wards in the Royal Infirmary there was bruising of the left forearm and back and a compound depressed fracture of the skull on the left side, the centre of the wound and of the depressed fragment being 9 cm. above the external auditory meatus and 1 cm. in front of this. The wound in the scalp was a linear one, running downwards and forwards, about 4 cm. long; the depressed fragment corresponded to the wound in length and direction. There was paralysis of the lower part of the right side of the face; the tongue was protruded to the right side, he spoke with great difficulty, articulation very slow and laboured, apparently from paralysis of lips and tongue, and he had considerable difficulty in swallowing. There was no apparent loss of power or of co-ordination in the right arm and hand, and there was no alteration in the sensory functions. The wound was enlarged and the depressed fragment removed. The dura was torn and bled freely. Several pieces of brain tissue lay in the wound and were removed. The wound was closed, and healed by first intention, the brain visibly pulsating through the tear.

The different motor paralyses recovered very slowly, there being no visible improvement until twelve days after the operation. On the third day after operation there was active twitching of the muscles at the angle of the mouth on the right side for two minutes.

On the fourth day there was first noticed distinct weakness of the right hand and diminished sensibility to heat, pain and touch. When the fingers were pricked with a pin he says it is hot. Co-ordination of right hand defective; he can't button his shirt, or pick up a pin, or point to his limbs, or make his forefingers meet with his eyes shut. Sensibility in face and arm normal. Fundus of eyes normal. Electrical reactions normal.

The sensory symptoms recovered by the 19th; motor paralysis recovered more slowly. The tongue was protruded straight on 23rd inst.

3. *Dr Alexander James* exhibited a man, aged 27, suffering from DOUBLE AORTIC AND DOUBLE MITRAL DISEASE. He had had several attacks of rheumatic fever, the first one, from which his cardiac mischief probably dated, had occurred fifteen years before. This patient's pulse was of the water-hammer character,

but Dr James pointed out that it presented also a character which he thought had not been sufficiently taken notice of by medical writers. This was what he called "predicrotism." This could be felt by the finger, and could be, as a rule, easily distinguished by the finger from dicrotism. The sphygmographic tracings, however, which he showed, illustrated its character best. As regards the cause of the predicrotic wave, a great many theories had been put forward. He did not intend to discuss these at present, but he showed cardiographic tracings from the same patient. The peculiarity in these tracings was that they showed a doubled ventricular systolic elevation. Indeed, the doubled ventricular contraction could be felt easily by the hand, and on auscultation a distinct doubling of the first sound could be made out. He stated that though he did not feel certain on the matter it seemed that the predicrotism might be due to asynchronism in the ventricular contractions. It was possible that one ventricle in contracting got a "shog" from the contraction of the other ventricle.

4. *Dr George Elder* exhibited—

(*a*) A CASE OF CONGENITAL ABSENCE OF THE RADIUS AND THUMB in a girl aged 9 months. As a result of this deficiency the hand was flexed at the wrist to an angle less than a right angle, and was also rotated somewhat towards the dorsal aspect; the outer side of the hand lying against the forearm. The ulna was considerably bent, as shown by a skiagram, but the bones of the carpus did not give a sufficiently strong outline to show which ones were absent.

The lobe of the ear on that side was deficient, and the thumb of the right hand was very small and in a fixed position of flexion into the palm.

There were no other deformities, and none of the other children or of the relatives had any malformations so far as could be determined.

(*b*) A CASE OF RHEUMATOID ARTHRITIS in a girl aged 16. The condition came on when the girl was about 5. She had never had any signs of rheumatism—no endocarditis, chorea, subcutaneous nodules, skin eruptions, or tendency to sore throats. Nor was there any rheumatic history in the family. For the last seven or eight years she had been very much crippled; the wrists, elbows, shoulders, ankles, knees, hips, joints of the

hands and feet, and cervical vertebrae were all much affected. Skiagrams showed that there were osteophytic outgrowths at the sides of the joints, though these were much less than one would have expected from the deformity of the joints. There was no enlargement of the spleen; whilst the only glands enlarged were those of the neck and axillae, and these only slightly, possibly from irritation of the head from pediculi. The fingers had a marked tendency to flexion to the radial side. The child had been brought up in poor circumstances.

It was evidently a case of true rheumatoid arthritis, as shown by the osteophytic outgrowths, grating on movement of the joints, absence of enlargement of the spleen or of the lymphatic glands generally, and absence of any of the signs of rheumatism in childhood.

5. *Dr J. O. Affleck* exhibited two patients who had been treated surgically for TUBERCULAR PERITONITIS. The first case was that of a man, aged 29, who was admitted into the Infirmary three years ago suffering from well-marked symptoms of tubercle both in the lungs and peritoneum. He had double pleurisy with effusion, for which he was aspirated, and there was in addition evidence of tubercular disease of the apex of the left lung. The abdomen was much distended with fluid effusion into the peritoneal cavity, and there was considerable pain and tenderness. Mr Duncan saw the patient and agreed in the diagnosis and in the proposal to treat the abdominal condition surgically. The abdomen was accordingly opened by Mr Duncan and a large quantity of fluid removed. A drainage tube was left in only for a day. The patient made an excellent recovery. The abdominal symptoms entirely disappeared, and although there remained the evidence of disease at the left apex it appears to have become quiescent, as the patient has been able to continue at his work as a printer since he left the Infirmary nearly three years ago, and his weight keeps up to its average.

The second case was that of a lad of 17, who is still under treatment and in whom the symptoms and evidences of TUBERCULAR PERITONITIS were well marked, but where no pulmonary complication existed. The abdomen was greatly enlarged and fluctuation could be easily made out. Mr Cotterill operated in the usual way, and a large quantity of fluid was evacuated.

The patient has gone on well, the temperature has come down to normal, and although he still looks very delicate he states that he feels greatly improved in every way.

Dr Affleck mentioned that he had had during the past six years nearly twenty cases of tubercular peritonitis treated in this way, the greater number of which, so far as he had been able to trace them, had done well. Four, at least, had proved unsuccessful, but there could be no doubt that in suitable cases, and especially where there was no evidence of intestinal ulceration with diarrhœa, an encouraging amount of success attended the surgical treatment of this disease.

The first of Dr Affleck's patients who had been treated in this way was a boy of 14, who was admitted into his ward in the Royal Infirmary in 1892. Mr Duncan operated with the most satisfactory results. The patient was at present serving as a soldier in Malta.

7. *Mr C. W. Cathcart* exhibited—

(*a*) A girl, aged 2½ years, AFTER OPERATION FOR INTUSSUSCEPTION. She had been sent into the Infirmary on November 26th, 1897, with an intussusception which could be felt at the anus. Its presence had been diagnosed four or five days before, but it had apparently been reduced by rectal injections several times, although it had returned on each occasion. When admitted the child was collapsed, and had been vomiting continuously for some hours. The abdomen was very tightly distended.

Under chloroform another attempt was made to reduce the intussusception by rectal injection, but without success, and the abdomen was then opened to the left of the middle line. The intussusception, which involved the great intestine only, was so firmly adherent that it could not be withdrawn by manipulation, and the enclosing bowel threatened to tear with the handling. The great intestine above the obstruction was then drawn into the wound, and an artificial anus made with a Paul's tube, the rest of the abdominal wound being sewn up. At the end of a week, when the Paul's tube had been removed, the wound suppurated, and as the child was crying and coughing a good deal the edges separated, and coils of small intestine covered with lymph were exposed below. They did not protrude, however, and the wound was gradually drawn together

with sticking plaster. Meanwhile, however, the intussusception had been gradually protruded at the anus, in spite of repeated attempts to push it back, and its presence there caused tenesmus and almost constant rectal irritation. It showed no signs of sloughing, but the efforts of the colon and rectum to expel it began to tell upon the bowel protruded at the artificial anus. The loop of bowel was diminished greatly in size, and there was a fear of the opened part being dragged inside the peritoneal cavity. A second operation was therefore undertaken on December 17th, in order to remove the mass of intussuscepted bowel. This was effected through the wall of the colon by a second opening in the abdominal wall below and to the left of the first. The cut edges of the colon were united to those of the skin, thus making a second artificial anus.

On January 21st the original artificial anus was closed, and the second artificial anus was similarly treated on the 8th of February. Mr Cathcart was much indebted for valuable advice in the operations for closing the artificial anus to Dr John Duncan, whose method he followed, and for help in planning out the operation for excision of the intussuscepted bowel.

(*b*) A patient AFTER OPERATION FOR RUPTURED TUBAL PREGNANCY. Mrs M., æt. 30, woke up about 3 a.m. on Saturday, January 1, 1898, suffering from intense pain all over the abdomen. This was followed shortly by vomiting, which continued all day whenever she tried to take anything by the mouth. Dr James Smith was sent for, and suspected the possibility of appendicitis. The pain continued in spite of soothing remedies, and as the patient's strength began to fail rapidly on Sunday Mr Cathcart was asked to see her in the evening. She was collapsed and had a very feeble pulse of 130 beats per minute, with a subnormal temperature. Her abdomen showed no respiratory movements, and felt very firm. It was tender to the touch all over but especially to the right of and below the umbilicus, *i.e.*, in the region of the appendix. The patient had not been pregnant for several years. She had missed the last two periods and had had morning sickness, but as her menstruation had been irregular for some time she had not considered herself to be certainly pregnant. During the week or ten days which preceded the attack she had felt pain from time to time in the place where it was afterwards most severe. The diagnosis lay between ruptured tubal pregnancy and a

ruptured vermiform appendix, and immediate operation was recommended.

An oblique incision was made over the region of the appendix and the layers of the abdominal wall were split.

The peritoneal cavity contained blood and in the extremity of the right Fallopian tube an ovum about the size of a Tangerine orange was found still in position although its containing sac had given way. It was lifted out and the distended Fallopian tube was then ligatured with silk and cut off close to the uterus. Masses of dark clot were then removed from the pelvis, and more than a pint measure of them was afterwards collected. The pelvic cavity was washed out with boiled water and the wound was sewn up without drainage.

The patient made a slow recovery, but was now in excellent health with the wound soundly healed.

The shaggy chorion containing the fœtus, about an inch in length, and the distended Fallopian tube were shown to the meeting ; preserved by Jores' method.

8. *Dr G. A. Gibson* exhibited—

(*a*) A patient with CHRONIC SYPHILITIC DISEASE OF THE NERVOUS SYSTEM. The patient was a married woman, aged 36, with no specific history, but a series of repeated miscarriages. She suffered from lightning pains, girdle sensations, and gastric crises. The Argyll Robertson phenomenon was present. The legs were feeble, the myotatic irritability abolished, and the control of the ophinetus lost. There was no loss of the muscular sense, and co-ordination was intact. Advanced choroiditis was present in both eyes. The case was regarded as one of pseudo-tabes syphilitica.

(*b*) A woman, aged 30, presenting physical signs absolutely conclusive of the diagnosis of obstruction and incompetence, both mitral and tricuspid. There were two distinct presystolic murmurs, one at the apex, the other at the left external edge. The cause was obscure.

9. *Dr James Carmichael* exhibited a boy, aged 9, suffering from PROGRESSIVE MUSCULAR SCLEROSIS. There was well marked hypertrophy of the calf muscles and the deltoids, with atrophy of the latissimus dorsi, supra and infra spinatus. His

gait was characteristic and also the "climbing up" in rising. Two brothers were affected in the same way.

II. Exhibition of Photographs, &c.

Dr Dawson Turner exhibited some Stereoscopic Röntgen Photographs of the injected blood vessels and an experiment of Mr Shelford Bedwell's on a subjective colour phenomenon.

III. Exhibition of Specimens

1. *Dr C. W. MacGillivray* exhibited A UTERUS AND APPENDAGES which he had removed for large fibro-myomata from a woman, æt. 50, in his wards in the Royal Infirmary.

As she had suffered acutely for many years, the tumours entirely blocking up the upper pelvic opening, and their causing her great pain, constipation and inability for all work, Dr Macgillivray thought it right to accede to her request for an operation. The method employed was the median abdominal incision. The condition of the patient after the operation never gave rise to the least anxiety, and she made an excellent recovery.

2. *Mr C. W. Cathcart* exhibited A SPECIMEN OF MULTIPLE GASTRIC ULCER. The patient, a young woman, aged 24, had had an attack of hæmatemesis in November 1897. She had recovered and was apparently in excellent health, with no gastric symptoms, when she was again taken ill.

The attack came on suddenly on the evening of Monday, April 25, 1898. She had severe abdominal pain and vomiting with some hæmatemesis. She was sent into the Infirmary on the evening of the 27th. The pain had somewhat abated—partly owing probably to morphia—but she had a raised temperature, a pulse of 120, a tympanitic note over the liver, and all the local symptoms of severe peritonitis. The abdomen was opened without delay. A small perforation was found on the anterior surface of the stomach. Its edges were too soft to hold a thread, but a fold of the healthy stomach wall was stitched over it. The peritoneal cavity was then thoroughly douched out, both near the stomach and in the pelvis, and drainage was established in both places.

For four days the patient's progress was quite satisfactory.

On the fifth morning the pulse was noticed to be very rapid, and in the evening she became collapsed. She grew weaker, and died on the afternoon of the sixth day after the operation.

At the *post-mortem* examination it was found that she had died from hæmorrhage and extravasation into the peritoneal cavity of altered blood, which had been caused by a large ulcer on the posterior surface of the stomach. The blood from this ulcer had been blackened by the action of the gastric juice, and had then partly passed down into the small intestine which contained a large quantity of it, and partly had escaped into the lesser sac of peritoneum through this second ulcer. After filling this cavity it had overflowed through the foramen of Winslow into the right hypochondrium. Besides this one, a third ulcer near the pylorus adhered to the liver, and a fourth near the one which had been operated on was just beginning. The fold of stomach still occluded the ulcer which had perforated.

The absence of symptoms from these ulcers was noteworthy. The mucous membrane seemed otherwise healthy.

3. *Dr G. A. Gibson* exhibited—

(*a*) A LARGE GALL-STONE REMOVED FROM THE GALL-BLADDER by Mr Caird. It was on section found to be composed of cholesterine, with a core of inspissated bile. The gall-bladder was full of sterile pus, and the cystic duct closed.

(*b*) A WATER-COLOUR DRAWING FROM A CASE OF ADDISON'S DISEASE. In spite of supra-renal treatment death ensued, and at the autopsy the adrenals were found in a caseous condition, as shown by Dr Fleming.

(*c*) A PHOTOGRAPH, taken with the X rays by Dr John Macintyre of Glasgow, from a case of complete transposition of the viscera, which had been kindly sent to him by Dr Cattanach.

4. *Dr Robert A. Fleming* exhibited MICROSCOPIC AND NAKED-EYE SPECIMENS FROM THE CASE OF ADDISON'S DISEASE referred to by Dr George A. Gibson (see page 184). The naked-eye specimens included the two supra-renals, skin from pubic region, mucous membrane of mouth, and a "mole" from the back. The microscopic specimens shown were sections of both supra-renals, sections of skin showing the great increase of pigment in the cells of the Rete Malpighii, and a section of one of the "moles."

Dr Fleming remarked that the supra-renals were almost entirely caseous, the cutical portion being firmer and more cartilaginous than the medullary. A few giant cells were found.

The semilunar ganglia were apparently unaffected. In contrast with this case, Dr Fleming showed two supra-renals from a male, aged 34, which were markedly affected by tubercular deposit, but in that case extremely little pigmentation of the skin had resulted. One essential difference between the supra-renals of the two cases was, that in the second case much supra-renal tissue was left unaffected. Both cases were unquestionably of tubercular nature.

Meeting IX.—June 1, 1898

DR JAMES CARMICHAEL, *Vice-President, in the Chair*

I. ELECTION OF MEMBERS

D. G. Macleod Munro, M.B., C.M., Alloa, was elected an Ordinary Member.

II. EXHIBITION OF PATIENTS

1. *Dr Norman Walker* exhibited—

(*a*) A case of LICHEN SCROPHULOSORUM occurring in a child who had tubercular joint disease. The papules and their distribution were both exceedingly characteristic, but had lost somewhat in appearance during the week the case had been under treatment with cod liver oil. The case he showed a few months previously had disappeared under the same treatment in a few weeks.

(*b*) A case of FOLLICULITIS DECALVANS (?). A little girl, a patient of Dr M'Bride's, had, when he saw her, a patch of complete baldness looking to superficial observation like Alopecia areata. The border however was red and granular looking and a fluid was exuding from it. In the immediate neighbourhood was another patch weeping profusely, and apparently indicating the first stage of the affection. The hairs when stained were found to be sheathed with micrococci,

which resembled those described by Quinquad. Treatment seemed to have little effect, the disease extending slowly. The hair was exhibited under the microscope.

III. Exhibition of Specimens

1. *Dr Norman Walker* exhibited sections from a case of Lupus erythematosus of the Cheek.

2. *Dr Alexander Bruce* exhibited—

(*a*) A life-size cast and photograph of a case of achondroplasia which had been under his care. The patient, a man of 40 years of age, was married, and the father of two healthy, well-grown children, who showed no physical defect He himself was born of healthy parents and none of the collateral branches of the family, so far as he was aware, showed any tendency to a similar deformity. His height was 46 inches, and the diminutive stature was due to the great shortness of the lower limbs rather than to any diminution in the length of the vertebral column. Both the upper and lower extremities were very short and the long bones were remarkably thick, being straight but considerably enlarged at the extremities, especially the ankle and wrist. The hands were very broad; the fingers short and broad. The first and second fingers showed a tendency to deviate to the radial side, the third and fourth to the ulnar side. The nails on both the fingers and toes were remarkably short. A skiagraph of the hands made by Dr Harry Rainy showed that the proximal phalanges were thickened to a remarkable degree. The remaining phalanges and metacarpals were affected to a slighter extent. The bones of the carpus were large, and some of them, more especially the scaphoid, were abnormal in shape. The mental development was such that the patient had at one time been able to act as a teacher in a primary school in a remote district in Westmoreland. The head was well formed, and the face had a dignified aspect which contrasted oddly with the puny body and feeble ungainly gait of the patient.

(*b*) Sections from the basal ganglia and medulla of two individuals who had suffered from attacks of hemiplegia due to limited hæmorrhages. The sections demonstrated that these hæmorrhages, which were of small size and which had riddled the basal ganglia with small cavities resembling those found

in Gruyère cheese had arisen in arteries which were the subject, not of miliary aneurism, but of arterial sclerosis, or chronic senile obliterative arteritis. The hæmorrhages had occurred at points where the wall of the vessel had become thinned, or from minute blood-vessels in the adventitia. The hæmorrhage had in every instance distended the peri-vascular sheath at first and subsequently passed into the brain substance. The point which it was desired to emphasise was the very important part played in senile hæmorrhage and in senile cerebral softening by the gradual thickening of the inner coat of the vessels.

(*c*) A series of sections made from the various organs in the rare disease "diabète bronzé" which has been observed on the Continent, but, so far as he is aware, not yet met with in Britain. The sections were cut from portions of tissue presented to him by Dr Pierre Marie of Paris. In the disease, which is characterised by diabetes, enlargement of the liver, bronzing of the skin and a progressive anæmia, there is a condition of hypertrophic cirrhosis of the liver with a remarkable degree of pigmentary degeneration in the liver cells and a pigmentary deposit in the trabeculae of fibrous tissue separating the liver-cells. The pigment granules in these situations give a prussian blue reaction with ferrocyanide of potassium and hydrochloric acid. Similar pigmentary deposits were found in the heart muscle and in the spleen. Sections from all these organs were shown.

3. *Dr Harvey Littlejohn* exhibited—

(*a*) AN ANEURISM OF LEFT VENTRICLE OF THE HEART. The anterior ventricular wall was dilated and occupied by an organised clot the size of a crown piece. Microscopic sections showed that the muscular tissue had almost disappeared under the clot, and that the walls of the arteries were very much thickened. The patient, æt. 56, had complained of pain over the heart, and shortness of breath, but had been working and was comparatively well the day before her death. She was found dead in bed.

(*b*) A case of SUICIDAL CUT THROAT. The position of the wound was unusual, being at the level of the trachea, which was almost completely divided in two places at its upper end. The jugular vein and common carotid on the right side was divided. The vessels on the left side were uninjured.

(*c*) An interstitial extra uterine pregnancy. The woman, married, æt. 30, was seized with symptoms which were regarded as indicating an attack of cholera, and died in the course of twenty-four hours. The *post-mortem* revealed a tumour about the size of a small marble on the left side of the uterus near the fundus, which had ruptured and caused a large effusion of blood into the pelvic and abdominal cavities. The pregnancy appeared to have existed for about four weeks.

(*d*) Preparation showing an old cicatrized suicidal cut-throat wound of the neck and larynx and the cicatrix of the tracheotomy wound made for its treatment.

(*e*) Preparation showing the cicatrix left in the larynx by an old tracheotomy wound, and partial stenosis of the trachea resulting from the operation.

IV. Original Communications

I. PERSONAL EXPERIENCES IN THE TREATMENT OF ENLARGED PROSTATE

By ALEXIS THOMSON, M.D., F.R.C.S.Ed., Assistant Surgeon to the Royal Infirmary and Lecturer on Surgery, Edinburgh

In view of what has been written on this subject it seems undesirable to attempt to review the subject as a whole; I shall, therefore, restrict myself to personal experiences, as indicated in the title of this paper.

From a large number of cases of senile enlargement of the prostate, which have been under my care during recent years, I propose to communicate the particulars of six in which a "curative" operation was performed. It is, of course, impossible, that any valid conclusions can be drawn from so small a number, but it seems to me to be the duty of everyone to record his own experience in the treatment of a disease, regarding which there exists so much difference of opinion. It may be stated at the outset that cases in which the patient pursues the even tenor of his life under the ægis of the catheter, are excluded from the present inquiry. We are here concerned with those in which, after the existence of prostatic symptoms for a varied period of month or years, with or without the establishment of catheter life, the patient is drifting into a condition of illness and danger, with increasing residual urine,

increasing difficulty and pain in the use of the catheter, and it may be the existence of complications, *e.g.* cystitis, false passages, hæmaturia, orchitis, and constant imminence of renal or of general septic infection. Prostatic cases are not uncommonly in this condition when they are first brought under the observation of the surgeon. Until recent years it was the custom for him to aim at tiding the patient over the immediate crisis, by what may be described as medical measures, along with skilful catheterisation at intervals, or continuous, resorting only in the more serious cases to operative drainage of the bladder, as introduced and practised by Annandale and Harrison. At the same time it was well known that a recurrence of the crisis was to be anticipated sooner or later and upon repeated occasions, until finally the patient would fall a victim to the long anticipated urinary infection ; hence the dictum came to be, if a patient with a badly behaved enlarged prostate lived long enough, *i.e.* did not succumb to some other lesion, he would die of surgical kidneys or of septicæmia.

While gratifying results have been obtained from drainage of the bladder, by Annandale, Harrison, and others, the method is not applicable in anything like a large proportion of the cases which present themselves, either in hospital or private practice ; so much depends upon the continuous supervision of the fistula and of the necessary apparatus, both on the part of the patient and of the surgeon, that even where established successfully, there is still the risk of infection and of a recurrence of the crisis, similar to that which attends the failure of catheter life, so that the procedure in question is practically reserved as a palliative measure, for cases in which there already exist evidences of grave septic infection, while at the same time the possession of a vesical fistula cannot be regarded as an ideal state of affairs. Surgical treatment in the majority of cases should aim at what may be described as a radical cure, *i.e.* at eliminating the obstructing and progressively enlarging prostate, and in this way freeing the patient from the absolute sway of the catheter, and at restoring the functions of the bladder, so far as this may be possible.

Bottini's operation, which consists in destroying or in dividing the obstructing prostatic tissue by the galvano-cautery, has been practised by its author for the last twenty-two years. He has had only two deaths in a series of eighty cases. The procedure

was brought before the Edinburgh Medical-Chirurgical Society some years ago by a distinguished assistant of Bottini, but, so far as I am aware, it has been little practised in this country. Freudenberg, of Berlin, has employed it extensively, and corroborates all that has been claimed for it by Bottini. The prejudice with which it is regarded by many, results from the difficulty in safely regulating the operation, until considerable experience has been acquired in the manipulations of the instruments employed.

Suprapubic prostatectomy is rightly regarded as the most certain curative procedure, where the difficulty in emptying the bladder depends upon an intravesical projection of the prostate, and especially where there is evidence of the existence of a pedunculated middle lobe, which may lend itself to removal with the scissors or tumour forceps. The risk of the operation would not appear to be appreciably greater than a simple suprapubic cystotomy, and is not a serious one, in the absence of already existing septic infections of the bladder and kidneys. Only one case has come under my care in which this procedure was indicated ; one in which a stone lay behind a projecting middle lobe. The particulars are briefly as follows :

A vigorous schoolmaster, æt. 57, was sent to me by Dr Peterkin, of Forfar, with symptoms of stone in the bladder. Twenty-two years before, he had passed a small calculus by the urethra. His present ailment was of twelve months' duration, and was characterised by frequency of micturition and hæmaturia, both aggravated by exercise or jolting. He had had, at any rate, one attack of retention, during which the urine had to be withdrawn thrice daily. On admission to the Deaconess Hospital, he was able to pass an ounce or two of water, at frequent intervals. The urine was acid and deposited abundant mucus and pus. The sound, with its beak rotated downwards, struck a rough fair-sized stone lying on the floor of the bladder. A soft catheter had to be introduced nearly 11 in. before urine escaped. Per rectum, there was apparently a very moderate degree of general enlargement of the prostate. The existence of a projecting middle lobe was inferred (1) as the most probable explanation of his dysuria ; in the absence of general prostatic enlargement ; (2) from the increased length of catheter required to evacuate urine ; and (3) the manipulations of the sound, required to strike the stone. The amount of residual

urine varied from 5 to 15 oz. In November 1895 I opened the bladder by a horizontal incision above the pubes ; the stone was found lying behind a semipedunculated middle lobe, of the size of a large cherry, and was easily removed ; the middle lobe was then made to project by two fingers introduced per rectum, and its neck torn through with the angled tumour forceps. Sutures were passed through the edges of the wound in the bladder, before the latter was irrigated with hot lotion (boracic), as I had found in a case of ruptured bladder that the contractions caused by the hot fluid may render accurate stitching very difficult. A catheter was tied in the bladder, and gave exit during the next twenty-four hours to a large amount of bloody urine. Thereafter, the urine escaped from the wound above the pubes, apparently in the first instance because the catheter became repeatedly blocked with clot, and the bladder was allowed to become over-distended ; a little later the sutures gave way altogether.

The entire wound opened out and became the seat of a sloughing cellulitis, which caused a protracted illness, and prevented the wound closing for several weeks. During this time the urine was turbid and slimy, he was taking sandal-wood oil and salol in full doses. Four months from the date of operation he wrote that he was making water as easily as ever he did in his life, every three hours during the day, and twice or thrice during the night, and that it was quite healthy ; that he had passed small calculi by the urethra on several occasions ; these calculi were apparently derived from the kidneys, chiefly from the right, judging from the unilateral colic. His diet may have been partly to blame for the continued lithiasis, for, according to his own statement, the only thing he never tired of was brandered steak, and that he was in the habit of eating three times in the day. Six months from the operation he had only to rise once in the night, and even that was unnecessary, while during the day he usually made water every four hours or so. Three months later he passed another small calculus, after repeated slight attacks of left renal colic. In March of this year (two years and five months after operation) he succeeded in passing a uric stone, 1 inch in length and nearly $\frac{1}{2}$ inch in breadth ; this caused severe colic and evanescent hæmaturia, while passing down the left ureter ; it remained eight days in the bladder with occasional abortive attempts to enter the

urethra ; finally, it entered the pipe, on suddenly rising from his chair, and was expelled without much difficulty. Apart from these attacks of lithiasis, he is perfectly well, teaching in the school, golfing, and making his water easily at normal intervals.

Here, then, we have a case of projecting middle lobe without any existing septic infection ; an ideal one for suprapubic prostatectomy ; the ultimate results of the operation performed two years and six months ago are all that could be desired, the urinary functions being performed normally, and the outlet for the bladder, instead of being obstructed, has permitted of the escape of calculi, one of which, at anyrate, would have had difficulty in entering the normal prostatic urethra.

The following cases, in which a sexual operation was performed, were all examples of well-marked general prostatic enlargement, in which the disease had reached a critical stage, in virtue of the failure of catheter life and of the development of complications, rendering some curative operations necessary, in order to prolong if not preserve life. Four of these were subjected to double castration, of which the earliest was operated upon two years and eight months ago ; the fifth case was a patient with only one testicle, the *vas deferens* of which I resected thirteen months ago. The particulars are briefly as follows :—

Case 1.—*Cases of Double Castration.*—J. B., æt. 68, recommended to Professor Annandale's wards by Dr Graham, of Currie. Frequency of micturition, twelve months ; one previous attack of retention ; has to make water every half-hour during the day, and every two or three hours during the night, and the act is attended with burning pain ; he can pass catheter but it pains him, and frequently causes bleeding. The urine is neutral and contains pus and albumin. The residual urine amounts to 5 to 10 oz. The prostate, as felt per rectum, has a breadth equal to three fingers. The top of a long index finger can just reach its upper border.

Double castration, 6th August 1895.—Three weeks later, he was making water much more easily and at longer intervals, and the residual urine was reduced to 2 oz. He now enjoys a good night's rest, which he has not done for twelve months. Four months later he was so well that he sailed for Melbourne to visit his family. He was there visited, eight months later

(twelve months after operation) by Dr James Laing, a former house surgeon to Professor Annandale, who reported as follows: He had gained 21 lb. in weight, and was in the best of health. No enlargement of the prostate could be felt per rectum. He had not used a catheter for three months. He passes water every two and a half hours during the day, and as a rule four times during the night; the latter frequency he attributed to sleeplessness, as there is no real desire to micturate, but as he lies awake he has got into the habit of rising and emptying the bladder. Pus has disappeared from the urine; its specific gravity is 1018, and contains only a trace of albumin. Unfortunately, a catheter was not passed, so there is no information as to the persistence of residual urine.

CASE 2.—*Double Castration.*—J. S., æt. 67.—Seen with Dr George W. Balfour and Dr Helen. Frequent and difficult micturition, four years; catheter life, one year, and on the whole satisfactory, apart from occasional attacks of urethritis. A recent and acute attack of right-sided epididymitis and hydrocele. The urine contained a few pus cells; the prostate generally enlarged, bulging into the rectum, and soft in consistence.

Double castration, 8th August 1895.—The wounds healed at once, and he was up at the end of a week.

Four months later his general health was excellent, and he was able then and since to ride to hounds. He uses the catheter thrice in the twenty-four hours; it passes very easily and gives him no trouble. Between times he makes water easily and when he likes, but does not empty the bladder, for if he uses the catheter after passing all he can he draws off 4 to 8 oz. His chief complaint is of sudden attacks of flushing and sweating, which are partly relieved by atropine and by camphoric acid. Two years and three months after operation, he writes that he only uses the catheter once in the twenty-four hours, just before going to bed, and that it does not give him any trouble.

CASE 3.—*Double Castration.*—J. N., æt. 69, seen with Dr Murdoch Brown, 22nd January 1896. Frequent, difficult, and painful micturition, two years; only passes a few ounces at a time; he has to rise thrice in the night; the urine is normal. There is great difficulty in passing a catheter because of straining; he can't do it himself at all. A soft rubber catheter enters 11½ in. before the urine flows in a feeble stream. Per

rectum the prostate is firm and very much enlarged. There is a recent hydrocele of the tunica vaginalis on both sides.

His life is rendered a burden by the dysuria, and catheter life is impossible.

Double castration, 2nd February 1896.—He was up at the end of a week. Since then he has maintained excellent health; has no pain nor any real annoyance connected with his bladder.

19th April 1898.—He only rises once in the night, and makes water quite easily, so that he gets a good night's rest. During the day he makes water every two or three hours, but thinks this is largely habit. Only uses the catheter (No. 8 gum elastic) when he has not had an opportunity of making water, and his bladder has become distended; it is passed quite easily; the urine is 1020 acid, and contains a few pus cells; there is only half an ounce of residual urine. The prostate is normal in size and in consistence.

CASE 4.—*Double Castration.*—J. W., æt. 76, seen with Dr Murray Lyon, Midcalder. Frequency of micturition, ten years. Retention for thirty-six hours, four years ago; since then catheter life, with, however, increasing difficulty and pain, latterly amounting to agony, both at the neck of the bladder and at the point of the penis, during and after the emptying of the bladder. On two occasions there has been considerable hæmaturia. He described his life as being a burden to him, on account of his sufferings day and night. His prostate is about the largest I ever felt, and is very firm in consistence. A moderate degree of hydrocele is present on both sides, and some urethral discharge.

Double castration, 29th November 1897.—In a few days he was able to pass 2 or 3 oz. at a time without the catheter; the latter was required thrice a day to empty the bladder; it is passed quite easily. He expresses himself as having entered a new lease of life; the chief benefit has been the relief of pain.

16th April 1898.—Catheter required as before; is easily passed and gives no trouble.

CASE 5.—*Unilateral Vasectomy in patient with only one testicle.*—J. S., æt. 75; seen with Dr Hunter of Linlithgow, 20th January 1897. Frequency of micturition, ten years; first attacks of retention, eight years ago; acute epididymitis

on left side, six years ago, resulting in complete atrophy of the testicle ; the epididymitis was apparently the result of a catheter urethritis. The atrophy of the one testicle had no apparent influence on his ailment. He had acquired the habit of straining at micturition, and the latter act was frequently attended by involuntary evacuation of the bowels. He could no longer pass a catheter, and he lost the power, more and more, of emptying his bladder, and merely passed little dribbles frequently. A week before I saw him the retention became complete, and he was aspirated above the pubes ; the large rectal prostatic catheter was then passed and tied in. This gave rise to painful spasms of the bladder and loss of sleep, and he began to go down hill rapidly.

When Dr Hunter and I saw him together, his condition was very critical. The prostate was firm, generally enlarged, and corresponded to the breadth of three fingers from side to side. Under chloroform the only instrument I could pass was the largest size silver one, of the pattern introduced by Professor Chiene. As we knew that he could not bear to have this catheter tied in, we considered it necessary to open his bladder above the pubes, and thus arrest the threatening septicæmia. The prostate was felt to project into the bladder as a firm, rounded mass, like a tangerine orange. Within three days he was sitting up in a chair, greatly improved in every respect. Towards the end of the third week the fistula was closing rapidly, when the bladder again became distended ; it was quite as difficult to pass a catheter as it had been before, and he was again passing into a critical state. I therefore re-opened the suprapubic wound, and passed a tube into the bladder, and at the same time (6th March 1897) *resected one inch of the vas deferens* of his remaining (right) testicle. He was soon up and going about, with a tube from the suprapubic opening, leading to a urinal inside the leg of his trousers. Three months after the vasectomy he was seen by Dr Crawford Renton, in Glasgow, who passed a No. 9 soft catheter quite easily.

29th April 1898. — Suprapubic fistula still relied upon and kept open ; occasionally a little by uretha. Prostate not appreciably altered. Some purulent discharge from the urethra.

Although the results in these four cases of double castration are neither very remarkable, nor all that could be desired,

Operation.	Age.	Duration of Symptoms.	Previous Retention.	Catheter Life.	Residual Urine.	Date of Operation.	Date of Improvement.	Result.	
								Prostate.	Bladder.
Suprapubic prostatectomy	57	One year	Once	...	5-15 oz.	Oct. 20, 1895	Three months later	No change	Normal micturition (passed calculi since)
Double Castration	68	One year	Once	Three months' pain, bleeding	5-10 oz.	Sept. 6, 1895	Three weeks later	Scarcely to be felt	Normal micturition (passed calculi since)
	69	Four ,,	?	One year, urethritis, epididymitis	10 oz.	,, 8, 1895	Four months ,,	?	Easy catheter, once in twenty-four hours
	69	Two ,,	Once	Impossible pain, orchitis	Bladder full and overflowing	Feb. 2, 1896	Three ,, ,,	Less than normal	Catheter only if distended, easy residual urine, ½oz.
	76	Ten ,,	Several	Four years' bleeding, recent agony, double hydrocele	Do.	Nov. 29, 1897	One week ,,	No change	Easy, painless, catheter twice daily
Vasectomy and suprapubic drainage	75	Ten years	Several	Several years, latterly impossible	Bladder full and overflowing	Mar. 6, 1897	Continues to rely on suprapubic fistula. Can pass catheter.		

still they are uniformly good. The operation is among the simplest in surgical practice; the patient need not be confined to bed for more than a few days. General anæsthesia is unnecessary. There were no evidences of any mental or bodily impairment which might be attributed to the loss of the testicles or to the abrogation of their functions. The urinary affection, which it was desired to benefit, has been robbed of its progressive and dangerous tendencies. Although the catheter is still required *as an adjunct* in three of them, it is no longer an absolute necessity; when required it is easily passed by the patient himself, without causing pain, hæmorrhage, orchitis, cystitis, &c. The contractile power of the bladder has improved, if it has not returned to the normal, and the residual urine has steadily diminished or disappeared. Their general health has greatly benefited, they have gained strength and weight, they enjoy their night's rest in a way to which they had long been strangers, and three of them have been able to resume their daily occupation. So far as their urinary organs are concerned, they give promise of enjoying continued good health. If it be borne in mind that every one of these patients had reached a critical stage of prostatic disease, which might be described as the beginning of the end, in which, under other circumstances, we should have given a bad prognosis, for it has been the experience that when catheter life fails the final cystic pyelo-nephritis or septicæmia is only a question of time, we may fairly infer, that had they been left without operation they might not be alive and well at the present day.

My own experience of vasectomy is limited, but, from the results published by others, it would appear to be inferior to castration, because its results are not only less rapid but less certain. Castration is, therefore, the operation of choice in the class of cases in which a sexual operation is indicated. It is to be rejected—(1) In cases where there are evidences of grave septic infection, for these can only be benefited by free drainage of the bladder; and (2) in cases where there exists an intravesical projection of the prostate, suitable for suprapubic prostatectomy.

2. RHEUMATIC MYOSITIS—SUBACUTE AND CHRONIC

By A. G. Miller, M.D., F.R.C.S.Ed., Consulting Surgeon to the Royal Infirmary, Edinburgh; Examiner in Clinical Surgery, University, Edinburgh

A great variety of affections is included under the term rheumatism. Among the public, and even in the profession, any painful condition is apt to be considered as rheumatic. In joint affections especially there is a wonderful vagueness about the use of the terms chronic rheumatism, rheumatoid arthritis, arthritis deformans, osteo-arthritis, and rheumatic gout. This is not to be wondered at, however, when one considers the difficulty that often exists of drawing a line between the various affections, and of making certain to which variety any given case belongs. Some affections have very distinct and characteristic symptoms—for example, acute rheumatism and acute gout. The subacute affections are more apt to be confounded—acute (so-called) tubercular arthritis, gonorrhœal arthritis, and rheumatoid arthritis. The chronic joint affections are, however, the ones most difficult to differentiate—chronic tubercular arthritis, gonorrhœal arthritis, and rheumatoid arthritis in their later stages, Charcot's joint, chronic traumatic arthritis, chronic rheumatism, and chronic gout.

Many cases are on the border line and not typical, and this variation may be due to constitution, inheritance, occupation, climate, diet, and other causes. I shall not venture into this subject, however tempting it may be. In the present paper I wish rather to draw attention to a few points in connection with subacute and chronic myositis.

I shall deal with the subject in the common and convenient arrangement of causes, symptoms, diagnosis, and treatment.

Causes

From careful observation and experience, I have come to the conclusion (which, of course, is not new), that the causes of rheumatism are cold, damp, heredity, and barometric influences. If we take myositis as an example, we find that it follows exposure to cold and damp, as when one gets wet fishing and has to travel home some distance in wet clothes. It is not the wetting that is to blame, for a person seldom gets harm from a wetting if he changes at once. Neither is cold

alone the cause, for most people can stand cold well enough provided it be dry. It is the combination of cold and wet (which one may call *chill*) that causes the rheumatism. It is possible that *evaporation* has something to do with the matter —letting wet clothes dry on one is well known as a cause of "chill."

But this is not all. Some people suffer while others escape, and it is well known that some must be more careful than others. The difference is, I believe, constitutional and hereditary, but it may be acquired. In these respects rheumatism resembles tubercle. It may, like the latter, be organismal, but, if so, it has the same peculiarity of finding some subjects a more easy prey than others. Again, there are certain barometric changes that seem to favour the occurrence of rheumatic affections, but I shall refer to this again in speaking of symptoms. I would sum up the etiology of rheumatism in this way—

The sufferer usually has a predisposing tendency (probably inherited) and has been exposed to both *cold* and *damp* before the attack comes on ; afterwards he is peculiarly susceptible to chills and barometric changes.

Symptoms and Diagnosis, Subacute Rheumatic Myositis

This condition is not unfrequently, I think, mistaken for articular rheumatism, especially when the muscle overlies a joint, as in the case of the deltoid. There is the usual history of rheumatic or barometric susceptibility, and of exposure to a chill as an exciting cause. Pain is present and often very acute. There is swelling, though not so much as in synovitis. There is also some fever and constitutional disturbance.

The two points which should guide the diagnosis are the situation of the pain and of the swelling. The pain is in the muscle itself. On pressure or percussion over the muscle pain is elicited, but more especially at the origin and insertion. Pain may also be produced by pinching the muscle. Movement causes pain, but not every movement. All voluntary contraction of the muscle is painful, and also those movements which stretch the affected muscle. Involuntary movements *which relax the muscle* do not cause pain, provided the patient does not resist.

Now as regards swelling. There is very little in myositis, while in synovitis there is considerable swelling from effusion into the joint.

I have noticed a symptom which, so far as I know, has not been specially described, namely this, that effusion takes place into the cellular tissue over the muscle, after the myositis has subsided somewhat. This effusion is not great, but gives rise to an audible crackling that can be elicited by moving the skin over the affected muscle. One may find this specially well marked in cases of intercostal rheumatism.

The myositis usually subsides in a few days, but is generally followed by a chronic condition which is very troublesome.

Chronic Myositis

Pain is here the prominent symptom. Pain of an aching character, more or less constant, worse at night, and varying with the weather. Pain may also be produced by pressure, and is aggravated by certain movements. There will also be stiffness and muscular wasting. The chronic affection is often the result of a subacute attack.

I think that the most characteristic symptom of chronic rheumatic conditions is the influence that barometric changes have on the pain. I have long studied this, and have come to the following conclusions :—

1. The pain comes on in advance of the change of weather. This experience is shared in by sufferers from stump neuromata and painful tubercle, and by some who have sustained fracture of a leg or arm.

2. The pain is often associated with a *falling* barometer, but may come on with a *rising* barometer if the wind be in the north or east. The pain is therefore not due to the falling of the barometer or diminution of atmospheric pressure, as some think.

3. The pain may subside when the rain has fairly set in, and a rheumatic person may be fairly comfortable even in a very wet day. It is evident, therefore, that the *rain* is not the cause of the pain.

4. Mists frequently cause rheumatic pains.

5. The advent of east winds may have the same effect even when they are not (apparently) associated with mist.

6. Pain may be conspicuously absent in cold, frosty weather, provided it be dry and bright.

My friend Mr W. S. Bruce, the naturalist, who has spent several seasons in the Arctic and Antarctic regions, assures me that neither he, nor anyone associated with him, has suffered

in the least from rheumatism, although exposed to the most intense cold for many months.

These experiences of Mr Bruce and others may point to an organismal cause for rheumatism, but they certainly prove that cold alone does not cause it.

There remains still the question of the barometric influence. How does it act, and why does the pain come on *before* the change of weather? If we could explain this premonitory barometric condition, we would, I think, get to the bottom of the whole matter.

I have only one suggestion to make—viz., that the pain is caused by a combination of cold and damp (as we saw at the beginning). I have frequently noticed a decided chilliness in the air preceding a shower, which passes off when the rain has fairly come on; and Mr Bruce has informed me that before rain the atmosphere may be more intensely saturated with moisture than when the rain is actually falling. This temporary chilliness I have also noticed to be associated with pain in my left shoulder, which is my rheumatic barometer.

Treatment

On this subject it is impossible to be dogmatic. One does not see much of results. Hospital patients seldom come back if they are *not* benefited, and still less frequently if they *are* benefited by treatment. First, as regards internal drugs, I believe that alkalies generally do good, and salicine and its salts are decidedly beneficial, especially in joint affections. Potassium iodide I have found useful mostly in nerve affections such as sciatica. An old remedy which has almost dropped out of use has sometimes a wonderful effect in muscular rheumatism. I refer to actea racemosa or cimicifuga. A full dose of the tincture (a drachm) I have seen remove the pain of lumbago or a stiff neck in a very short time.

Electricity I have tried, mostly in nerve rheumatism, and have found it relieve the pain and stiffness for a time. I do not think it has any curative effect.

In regard to local remedies, I give the first place to *heat*. As cold and damp cause rheumatic pains, so heat, especially dry heat, relieves. I do not say "cures"—for in my opinion nothing cures rheumatism, in this changeable climate of ours, in the sense of removing liability to relapses. Next to dry heat I would place counter-irritation. This action I would limit to

rubefaction, because by stopping short of vesication the counter-irritant can be repeated at will. Blisters, I think, are unsafe, because the pain which they cause induces the patient to keep the part at rest, and stiffness is apt to be the result. Liniments may be useful in many ways. If they contain an oily substance they protect the skin against cold ; if an anodyne, they relieve pain ; if an irritating substance, they may do good as counter-irritants ; and if they are well rubbed in, the friction may do good. If fomentations are employed they should be medicated. The best form is a solution of soda. I have seen rheumatic conditions disappear very quickly under alkaline fomentations.

Massage, in its widest sense, is very useful in chronic rheumatic affections. As pain is the principal symptom, and is best relieved by heat, so stiffness, next in importance, is most benefited by massage and movement. Movements must be both passive and voluntary. I have very little to say as to diet. I have not observed any influence exerted on rheumatism one way or another. Those patients who are benefited by dieting are, I suspect, gouty. It is not easy to differentiate between chronic rheumatism and chronic gout, and sometimes the two conditions manifest themselves in the same patient.

Climate is another subject on which I have not much to say. A dry and warm climate is best. I would sum up the therapeutics of rheumatism by saying that prevention is the most important thing. Persons who are " rheumatic " should take great care to protect themselves from cold and damp in every form.

In conclusion I would like to draw the attention of the members of this Society to the following points :—

1. That cases of myositis may be mistaken for articular rheumatism and treated by rest, which may result in stiffness from muscular contraction and atrophy.

2. That cases of apparent anchylosis from adhesions may be really the result of muscular contractions, and are more likely to be properly treated if this be recognised.

3. That cimicifuga is beneficial in rheumatic myositis. That it has no effect in acute articular rheumatism is no proof that it cannot benefit muscular rheumatism. In fact I am sure that I have proof to the contrary.

4. In the last place, I would like to have the opinion of members of this Society as to the probable explanation of the influence of weather changes on rheumatic pains ; for, were

this influence understood, much might be done in the way of prophylaxis.

I would like to say something on the diagnosis of chronic rheumatism from chronic gout. The two affections may exist together and even at the same time on the same unfortunate person. The two conditions are wonderfully similar. They are like twins that no one can distinguish from each other except when one sees them together. They are both hereditary. They both cause joint deformity and uselessness. They both occur mostly in advanced life. They both are the result, very frequently, of an acute or subacute attack. They differ mostly in the treatment necessary. Both are practically incurable, but the remedies that relieve are different in the two affections. They have also both to be differentiated from rheumatoid arthritis. I have tried to draw up a table that will represent the diagnostic points of each, and in doing this have availed myself of the writings of Drs Brabazon Bannatyne and Wollmann of Bath, Dr Reynolds, Dr Fortescue Fox, and Mr Arbuthnot Lane.

	Chronic Rheumatism	Rheumatoid Arthritis	Chronic Gout
1.	Disease of advanced life. Persons who have worked	Disease of early life; mostly females	Disease of advanced life; generally good eaters
2.	History of R. hereditary.	Sometimes history of acute rheumatism	History of gout—hereditary
	Tendency to barometric pains, etc.	No constitutional tendency to R. or G.	Tendency to gouty affections. Perhaps acute gout
3.	Otherwise healthy	Anæmia	Otherwise healthy
4.	Joints most affected = *fingers* symmetrical	Joints most affected = *fingers* symmetrical	Joints most affected = *fingers* asymmetrical
	Joints enlarged irregularly. Flexion-extension-flexion and ulnar deflexion	Joints spindle-shaped — fluid —lateral mobility	Joints enlarged — nodular uratic deposits
	Other joints affected = knees, hip, etc. Nerves and fibrous tissue affected	Several other joints affected (jaws and neck, Bannatyne), sweating of palms	Perhaps ulnar deflexion and flexion-extension-flexion
	Toes not affected	Toes not affected	Toes affected
5.	Muscular atrophy from want of use; improved by exercise	Muscular atrophy well-marked —special and general (interossei)	Muscular atrophy not marked
6.	Benefited by alkalies and warm applications	Not benefited by drugs but by hot air and baths—massage	Benefited by alkalies
	Aggravated by cold and damp and barometric changes	Aggravated by cold and damp and barometric changes	Not affected by cold and damp and barometric changes
	Temperature may be normal	Temp. 100 to 101	Temp. normal

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